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(FILE 'HOME' ENTERED AT 14:05:18 ON 10 JAN 2005)

FILE 'MEDLINE, EMBASE, BIOSIS, BIOTECHDS, SCISEARCH, HCAPLUS, NTIS, LIFESCI' ENTERED AT 14:05:42 ON 10 JAN 2005

L1 73150 S MYCOPLASMA
L2 63554 S NEURAMINIDASE? OR SIALIDASE?
L3 224 S L1 AND L2
L4 139 S (INFECTION? OR DISEASE?) AND L3
L5 2620647 S CANCER OR "HODGKIN'S" AND L4
L6 87 DUP REM L4 (52 DUPLICATES REMOVED)
L7 652 S (CANCER OR "HODGKIN'S") AND L6
E HIGUCHI M/AU
L8 3027 S E3
E SCHENKMAN S/AU
L9 344 S E3
L10 3371 S L8 OR L9
L11 2 S L6 AND L10
L12 8 S L6 AND (PREVENT OR INHIBIT)

=>

attenuation, and treatment of bacterial infections that may

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FILE 'LIFESCI' ENTERED AT 14:05:42 ON 10 JAN 2005

COPYRIGHT (C) 2005 Cambridge Scientific Abstracts (CSA)

=> s mycoplasma

L1 73150 MYCOPLASMA

=> s neuraminidase? or sialidase?

L2 63554 NEURAMINIDASE? OR SIALIDASE?

=> s l1 and l2

L3 224 L1 AND L2

=> s (infection? or disease?) and l3

3 FILES SEARCHED...

L4 139 (INFECTION? OR DISEASE?) AND L3

=> s cancer or "Hodgkin's" and l4

L5 2620647 CANCER OR "HODGKIN'S" AND L4

=> dup rem l4

PROCESSING COMPLETED FOR L4

L6 87 DUP REM L4 (52 DUPLICATES REMOVED)

=> s (cancer or "Hodgkin's") and l6

L7 652 (CANCER OR "HODGKIN'S") AND L6

=> d l6 1-87 ibib ab

L6 ANSWER 1 OF 87

MEDLINE on STN

DUPLICATE 1

ACCESSION NUMBER: 2004276433 MEDLINE

DOCUMENT NUMBER: PubMed ID: 15175306

TITLE: Spreading factors of **Mycoplasma** alligatoris, a
flesh-eating **mycoplasma**.

AUTHOR: Brown D R; Zacher L A; Farmerie W G

CORPORATE SOURCE: Department of Pathobiology, College of Veterinary Medicine,
University of Florida, Gainesville, FL 32611-0880, USA..
brownd@mail.vetmed.ufl.edu

CONTRACT NUMBER: 1R15HG02389-01A1 (NHGRI)
SOURCE: Journal of bacteriology, (2004 Jun) 186 (12) 3922-7.
Journal code: 2985120R. ISSN: 0021-9193.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200407
ENTRY DATE: Entered STN: 20040604
Last Updated on STN: 20040708
Entered Medline: 20040707

AB **Mycoplasma alligatoris** causes lethal invasive disease of alligators and caimans. A homolog of the nagH gene, encoding a hyaluronidase secreted by *Clostridium perfringens*, and a *C. perfringens* hyaluronidase nagI or nagK pseudogene were discovered in the *M. alligatoris* genome. The nagH gene was detected by PCR in the closest relative of *M. alligatoris*, *Mycoplasma crocodyli*, but not in 40 other species representing the *Mycoplasma* hominis, *Mycoplasma pneumoniae*, and *Spiroplasma* phylogenetic clusters. The hyaluronidase activity in the cellular fraction of *M. alligatoris* and *M. crocodyli* SP4 broth cultures was equivalent to 10(-16) U of *Streptomyces hyalurolyticus* hyaluronidase CFU(-1). Negligible activity was present in the cell-free supernatant fraction. No chondroitinase activity was detected. There is also a novel homolog of the nanI gene, which encodes a **sialidase** secreted by *C. perfringens*, in the *M. alligatoris* genome. The signature YRIP and SXDXGXTW motifs and catalytic residues of the clostridial **sialidase** are conserved in the mycoplasmal gene, but the leader sequence necessary for its secretion by *C. perfringens* is absent. The gene was not detected by PCR in any other *mycoplasma*. Potent cell-associated **sialidase** activity was present in *M. alligatoris* colonies on agar but not in the cell-free supernatants of broth cultures or in *M. crocodyli*. The presence of hyaluronidase and **sialidase** in *M. alligatoris* is consistent with the rapid invasiveness and necrotizing effects of this organism, and the lack of **sialidase** in *M. crocodyli* is consistent with its comparatively attenuated virulence. This genetic and biochemical evidence suggests that the spreading factors hyaluronidase and **sialidase**, a combination unprecedented in *mycoplasmas*, are the basis of the virulence of *M. alligatoris*.

L6 ANSWER 2 OF 87 EMBASE COPYRIGHT 2005 ELSEVIER INC. ALL RIGHTS RESERVED.
on STN

ACCESSION NUMBER: 2004515771 EMBASE
TITLE: Respiratory viral infections in high-risk patients.
AUTHOR: Greenberg S.B.
CORPORATE SOURCE: Dr. S.B. Greenberg, Baylor College of Medicine, Houston, TX, United States
SOURCE: American Journal of Respiratory and Critical Care Medicine, (1 Dec 2004) 170/11 (1142-1143).
Refs: 17
ISSN: 1073-449X CODEN: AJCMED
COUNTRY: United States
DOCUMENT TYPE: Journal; Editorial
FILE SEGMENT: 004 Microbiology
015 Chest Diseases, Thoracic Surgery and Tuberculosis
026 Immunology, Serology and Transplantation
037 Drug Literature Index
038 Adverse Reactions Titles
LANGUAGE: English

L6 ANSWER 3 OF 87 MEDLINE on STN DUPLICATE 2
ACCESSION NUMBER: 2004419768 IN-PROCESS
DOCUMENT NUMBER: PubMed ID: 15325005

TITLE: Trypanosoma cruzi trans-**sialidase** as a new therapeutic tool in the treatment of chronic inflammatory **diseases**: possible action against **mycoplasma** and chlamydia.

AUTHOR: de Lourdes Higuchi Maria

CORPORATE SOURCE: Pathology Laboratory, Heart Institute (InCor) of Clinical Hospital, School of Medicine of Sao Paulo University, Av. Dr Eneas de Carvalho Aguiar 44, 05403-000 Sao Paulo, SP, Brazil.. anplourdes@incor.usp.br

SOURCE: Medical hypotheses, (2004) 63 (4) 616-23.
Journal code: 7505668. ISSN: 0306-9877.

PUB. COUNTRY: Scotland: United Kingdom

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English

FILE SEGMENT: IN-PROCESS; NONINDEXED; Priority Journals

ENTRY DATE: Entered STN: 20040825
Last Updated on STN: 20041219

AB The present paper proposes a new therapy using Trypanosoma cruzi trans-**sialidase** to treat **diseases** with unclear pathogenesis that present in common chronic inflammation and fibrosis. This hypothesis is based on recent findings that co-**infection** with **mycoplasma** and chlamydia is present in many of these **diseases** and that this enzyme was capable to eliminate or decrease the co-**infection** from the host. We identified that **mycoplasmas** and chlamydias are present in atherosclerosis, aortic valve stenosis, dilated cardiomyopathy, chronic chagasic myocarditis and cancer. We hypothesized that mycoplasmal **infection** may induce immunodepression in the host, favoring proliferation of pre-existent chlamydial **infection** and that elimination of **mycoplasma** would lead to improvement of the immune system resistance and the control of chlamydial proliferation. **Mycoplasma** has a particular parasitic relationship with host cells, involving strong adherence of their membranes, making it extremely difficult to eradicate mycoplasmal **infection** from the host. A new therapeutic approach is suggested using one or more agents that prevent or inhibit the adherence of **mycoplasma** to host cell membranes by removing sialic acid residues and preventing oxidation of the cells. The use of a **neuraminidase** enzyme, particularly the T. cruzi trans-**sialidase** enzyme, associated with treatment using anti-oxidating agents is proposed. Preliminary experimental animal and laboratory tests showed good results. The proposal that trans-**sialidase** from T. cruzi is efficient in combating co-**infection** of **mycoplasma** and chlamydia is based, at least in part, on the observation that chagasic patients suffering from T. cruzi **infection** present less **mycoplasma** and chlamydia **infection** in their tissues. Also, a lower incidence of the **diseases** above described to be related to **mycoplasma infection** is observed in chagasic patients. It is also hypothesized that co-**infection** with **mycoplasma** and chlamydia may induce oxidation of the host cells. Anti-oxidants such as those present in plant extracts may also be used in the treatment. Other **diseases** such as chronic hepatitis, glomerulonephritis, Multiple Sclerosis, Alzheimer's Syndrome and idiopathic encephalitis are other examples of chronic **diseases** where **mycoplasma** and chlamydia might be present, as they have the characteristics of unknown etiology, persistent chronic inflammation and fibrosis.

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L6 ANSWER 4 OF 87 BIOTECHDS COPYRIGHT 2005 THE THOMSON CORP. on STN
DUPLICATE 3

ACCESSION NUMBER: 2003-27952 BIOTECHDS

TITLE: Composition useful for treating **mycoplasma infection** comprises an agent that prevents proliferation of **mycoplasma** or associated microbes;

native or recombinant enzyme treatment for **disease**
therapy

AUTHOR: HIGUCHI M D L
PATENT ASSIGNEE: HIGUCHI M D L
PATENT INFO: WO 2003082324 9 Oct 2003
APPLICATION INFO: WO 2003-BR49 28 Mar 2003
PRIORITY INFO: BR 2002-1010 28 Mar 2002; BR 2002-1010 28 Mar 2002
DOCUMENT TYPE: Patent
LANGUAGE: English
OTHER SOURCE: WPI: 2003-803968 [75]

AB DERWENT ABSTRACT:

NOVELTY - A composition comprises an agent (A) that prevents or inhibits the proliferation of at least one of **Mycoplasma** or microbes associated with **Mycoplasma**, is new.

DETAILED DESCRIPTION - An INDEPENDENT CLAIM is included for the use of an agent (A) for the manufacture of a medicament for treating a disorder defined by increased microbes proliferation associated with inflammation, fibrosis, calcification, ossification, cellular disarray and/or fragmentation of the extra-cellular matrix of the adjacent tissue.

ACTIVITY - Antimicrobial; Antibacterial; Antiinflammatory; Nephrotropic; Hepatotropic; Endocrine-Gen.; Cytostatic; Osteopathic; Antiarthritic; Antirheumatic; Gastrointestinal-Gen.; Cerebroprotective; Neuroprotective; Antiallergic; Vasotropic; Antiulcer; Respiratory-Gen.; Antiasthmatic; Virucide; Anti-HIV; Dermatological.

MECHANISM OF ACTION - **Mycoplasma** proliferation inhibitor; **Mycoplasma**-associated microbes proliferation inhibitor; Host cell proliferation inhibitor; Microbial proliferation inhibitor. Two rats presenting skin ulcer and tail injury due to the co-infection of Lycoplasma and Spirochetes were treated. One received 0.5 ml/animal TSN (complete active native trans-sialidase of Trypanosoma cruzi), every day for 10 days, and the other received TSC (active trans-sialidase substance catalytic portion, produced by a recombinant bacteria containing the Plasmodium (pTSIII), ATCC with PTA - 3483) for 8 days. The mice were killed respectively with 14 and 10 days. The skin ulcers already showed initial healing after 4 days of treatment, with complete healing in 14 days, with the formation of a new coat. There was a stop in the loss of the tail and the histological exam demonstrated regression of the lesion and severe decrease of all infectious agents.

USE - For treating or preventing **Mycoplasma** infection including disorders defined by co-infection and fusion of **Mycoplasma** and/or at least a second microbe to a host cell or a cell fragment, causing inflammation and at least one of the tissue alterations due to fibrosis, calcification, ossification, cellular disarray or fragmentation of the extra-cellular matrix of the subjacent tissue (e.g. aortic valve stenosis with calcification, idiopathic glomerulopathy, glomerulopathy with inflammation, Lyme's disease, co-infection with chlamydia, spirochete and/or archaea); and for the manufacture of a medicament for treating a disorder defined by increased microbes proliferation (e.g. calcification of the cardiac valves, glomerulonephritis, fibrosing chronic hepatopathy, baldness, and malignant neoplasia) (claimed). Also useful for the treatment of skin ulcer, osteoarthritis, inflammatory bowel disease, chronic cerebral sclerosis disease, lymphocytic chronic arteritis, non-purulent inflammatory osteoarthritis, multiple sclerosis, lymphocytic inflammatory vascular disease, optionally granulomatous and with non-stabilized etiology (e.g. Takayasu's disease, giant cell arteritis, Wegener's granulomatosis, thromboangiitis obliterans), rheumatoid arthritis, ulcerative colitis, Whipple's disease, gastritis, inflammatory diseases of the respiratory tract of not well established etiology (e.g. adult respiratory distress syndrome, Goodpasture's syndrome, asthma, chronic fibrosing hepatopathy, emphysema; and for the treatment or prevention of disorders associated with **mycoplasma** infection, co-infection and/or fusion of

mycoplasma with other microbes (e.g. virus such as human immunodeficiency virus, hepatitis virus, cytomegalovirus, human papillomavirus, Epstein-Barr virus; or bacteria).

ADMINISTRATION - The trans-**sialidase** enzyme is administered in a dosage of (4 mg/day) in a period of at least 2, or a culture of *Trypanosoma cruzi* with a mean trans-**sialidase** activity of 140 U/day is administered every other day for one week (1 - 8 weeks). The administration is intravenous, intraperitoneal, intrathecal, oral, by inhalation, subcutaneous or intramuscular.

ADVANTAGE - The composition inhibits or prevents the adhesion and/or infection of **Mycoplasma** and the microorganisms associated with them by at least 10%. The antibiotic protein such as **neuraminidase** enzyme or the trans-**sialidase** enzyme of *Trypanosoma cruzi* removes the sialic acid residues and inhibits or prevents the attachment of **Mycoplasma** to host cells.

EXAMPLE - No relevant example given. (24 pages)

L6 ANSWER 5 OF 87 BIOTECHDS COPYRIGHT 2005 THE THOMSON CORP. on STN
ACCESSION NUMBER: 2004-00309 BIOTECHDS

TITLE: Use of an agent that prevents or inhibits **Mycoplasma infection**, for manufacturing a medicament for treating or preventing a disorder associated with increased cell proliferation, e.g. atherosclerotic vascular disease or malignancy;
recombinant *Trypanosoma cruzi* protein application in infection, tumor and vascular disease therapy

AUTHOR: HIGUCHI M D L; SCHENKMAN S
PATENT ASSIGNEE: HIGUCHI M D L; SCHENKMAN S
PATENT INFO: US 2003124109 3 Jul 2003
APPLICATION INFO: US 2002-86913 1 Mar 2002
PRIORITY INFO: BR 2001-2648 3 Jul 2001; BR 2000-2989 3 Jul 2000
DOCUMENT TYPE: Patent
LANGUAGE: English
OTHER SOURCE: WPI: 2003-810968 [76]

AB DERWENT ABSTRACT:

NOVELTY - Use of an agent that prevents or inhibits **Mycoplasma infection** for manufacturing a medicament for treating a disorder associated with increased cell proliferation.

DETAILED DESCRIPTION - An INDEPENDENT CLAIM is included for a composition for treating or preventing **Mycoplasma infection** in a subject suffering from a disorder associated with increased cell proliferation or a co-infection with **mycoplasma** and a second microbe, comprising an agent that prevents or inhibits sialic acid-mediated attachment of **mycoplasma** to cells of the subject.

BIOTECHNOLOGY - Preferred Composition: The agent is an antibiotic or an enzyme having an activity consisting of **neuraminidase** and/or trans-**sialidase** activity. The enzyme is derived from a *Trypanosoma cruzi* microorganism, where the enzyme is a native or a recombinant enzyme. The enzyme has a fully defined sequence of 669 amino acids given in the specification. A vector containing the DNA insert having a fully defined sequence of 2010 bp given in the specification produces the enzyme.

ACTIVITY - Antibacterial; Antiarteriosclerotic; Cytostatic; Anti-HIV. A 64-year-old female patient with a palpable abdominal mass and a tumoral mass in the rectum was administered 50 ml of native trans-**sialidase** (TSN) intraperitoneally on alternate days for a period of 14 days. On day 23, with **mycoplasmas** confirmed in the bone marrow, erythromycin (500 mg/day) was given for a further 20 days. Clinical improvement and normalization of blood leukocytes was seen after 2 days. Considering the important clinical improvement and reduction in abdominal mass, a second session of TSN was administered. The patient demonstrated improvement in general clinical status. Tomography detected

a reduction in tumoral mass. Results showed that trans-sialidase is effective as a drug in the treatment of neoplasia, removing mycoplasmas from the neoplastic cells leading to their apoptosis.

MECHANISM OF ACTION - Neuraminidase; Trans-sialidase.

USE - The composition or the agent that prevents or inhibits mycoplasma infection is useful for manufacturing a medicament for treating or preventing a disorder associated with increased cell proliferation, e.g. atherosclerotic vascular disease or malignant disease, or a disease associated with co-infection with mycoplasma and a second microbe such as human immunodeficiency virus or a Chlamydia microbe (all claimed).

ADMINISTRATION - The amount of the enzyme administered is about 106-1013 units per day. Administration may be intravenous, intraperitoneal, intrathecal, oral, by inhalation, subcutaneous, or intramuscular. (32 pages)

L6 ANSWER 6 OF 87 HCAPLUS COPYRIGHT 2005 ACS on STN

ACCESSION NUMBER: 2003:261602 HCAPLUS

DOCUMENT NUMBER: 138:265609

TITLE: Use of neuraminidase inhibitors to prevent flu-associated bacterial infections

INVENTOR(S): McCullers, Jonathan A.

PATENT ASSIGNEE(S): St. Jude Children's Research Hospital, Inc., USA

SOURCE: PCT Int. Appl., 40 pp.

CODEN: PIXXD2

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2003026567	A2	20030403	WO 2002-US29417	20020917
WO 2003026567	A3	20040826		
W:	AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW			
RW:	GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG			
US 2004248825	A1	20041209	US 2004-809127	20040325
PRIORITY APPLN. INFO.:			US 2001-325615P	P 20010927
			WO 2002-US29417	A1 20020917

AB The invention provides a novel use for neuraminidase inhibitors in chemoprophylactic and treatment methods for the prevention, attenuation, and treatment of bacterial infections that may occur in association with, or as a sequelae of, viral influenza. The prophylactic methods of the invention are particularly suitable for the prevention of secondary bacterial infections, such as, but not limited to, infections of the lower respiratory tract (e.g., pneumonia), middle ear infections (e.g., otitis media), and bacterial sinusitis. The treatment methods are suitable for use in protocols designed to attenuate or treat bacterial infections that occur concurrent with, or as a sequelae of, the flu.

L6 ANSWER 7 OF 87 HCAPLUS COPYRIGHT 2005 ACS on STN

ACCESSION NUMBER: 2003:76631 HCAPLUS

DOCUMENT NUMBER: 138:135831

TITLE: Antibody heteropolymer complexes preparation and uses thereof
 INVENTOR(S): Taylor, Ronald P.; Craig, Maria L.; Hahn, Chang S.
 PATENT ASSIGNEE(S): University of Virginia Patent Foundation, USA
 SOURCE: PCT Int. Appl., 79 pp.
 CODEN: PIXXD2
 DOCUMENT TYPE: Patent
 LANGUAGE: English
 FAMILY ACC. NUM. COUNT: 1
 PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2003007971	A1	20030130	WO 2002-US23141	20020717
WO 2003007971	C2	20030410		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
EP 1416945	A1	20040512	EP 2002-770383	20020717
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, SK				
PRIORITY APPLN. INFO.:			US 2001-305989P	P 20010717
			WO 2002-US23141	W 20020717

AB The improved heteropolymer complex of the present invention comprises a first monoclonal antibody specific for a C3b-like receptor [complement receptor (CR1) or CD35 in primates and factor H in other mammals, e.g., dog, mouse, rat, pig, rabbit] site chemical crosslinked (covalently linked) to a second monoclonal antibody, in which the isotype of at least the second monoclonal antibody is the isotype having the highest affinity for the Fc receptor, e.g., in humans, IgG1 or IgG3. The invention also relates to methods for immune clearance of an antigen in a mammal via the C3b-like receptor comprising administering to said mammal an improved heteropolymer complex of the invention. Also presented are methods for treating or preventing viral **infection** or microbial **infection**, septic shock, or cancer, in a mammal comprising administering to said mammal an improved heteropolymer complex of the invention. The present invention further relates to pharmaceutical compns. for the treatment or prevention of the above **diseases** comprising an improved heteropolymer complex of the invention.

REFERENCE COUNT: 2 THERE ARE 2 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 8 OF 87 EMBASE COPYRIGHT 2005 ELSEVIER INC. ALL RIGHTS RESERVED.
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ACCESSION NUMBER: 2003235025 EMBASE
 TITLE: Neutrophil aggregates in a 13-year-old girl: A rare hematological phenomenon.
 AUTHOR: Claviez A.; Horst H.-A.; Santer R.; Suttorp M.
 CORPORATE SOURCE: A. Claviez, Department of Pediatrics, University of Kiel, Schwanenweg 20, 24105 Kiel, Germany.
 a.claviez@pediatrics.uni-kiel.de
 SOURCE: Annals of Hematology, (1 Apr 2003) 82/4 (251-253).
 Refs: 13
 ISSN: 0939-5555 CODEN: ANHEE8
 COUNTRY: Germany
 DOCUMENT TYPE: Journal; Article
 FILE SEGMENT: 004 Microbiology

010 Obstetrics and Gynecology
015 Chest Diseases, Thoracic Surgery and Tuberculosis
025 Hematology
037 Drug Literature Index

LANGUAGE: English

SUMMARY LANGUAGE: English

AB Aggregation of neutrophils in peripheral blood smears is a very rare, mostly self-limiting phenomenon and may result in pseudoleukopenia. In the majority of cases, malignancies, **infections**, or hepatic disorders have been identified as the underlying condition. Although the exact reason for neutrophil aggregation in vitro has not been clarified, its relation to the use of ethylenediamine-tetraacetate acid as an anticoagulant has been described in adults. We report here on the occurrence of transient neutrophil aggregation in a 13-year-old girl with Herpes simplex and concomitant *Mycoplasma pneumoniae* **infection**.

L6 ANSWER 9 OF 87 MEDLINE on STN DUPLICATE 4

ACCESSION NUMBER: 2004022687 MEDLINE

DOCUMENT NUMBER: PubMed ID: 14720004

TITLE: Treatment of community-acquired lower respiratory tract **infections** during pregnancy.

AUTHOR: Lim Wei Shen; Macfarlane John T; Colthorpe Charlotte L

CORPORATE SOURCE: Respiratory Infection Research Group, Respiratory Medicine, Nottingham City Hospital, Nottingham, UK..
wlim2@ncht.trents.nhs.uk

SOURCE: Am J Respir Med, (2003) 2 (3) 221-33. Ref: 116
Journal code: 101132974. ISSN: 1175-6365.

PUB. COUNTRY: New Zealand

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)

LANGUAGE: English

FILE SEGMENT: Priority Journals

ENTRY MONTH: 200402

ENTRY DATE: Entered STN: 20040115
Last Updated on STN: 20040224
Entered Medline: 20040223

AB The incidence of lower respiratory tract **infection** (LRTI) in women of child-bearing age is approximately 64 per 1000 population. The spectrum of illness ranges from acute bronchitis, which is very common, through influenza virus **infection** and exacerbations of underlying lung **disease**, to pneumonia, which, fortunately is uncommon (<1.5% LRTI), but can be severe. Acute bronchitis is generally mild, self-limiting and usually does not require antibacterial therapy. Influenza virus **infection** in pregnant women has been recently related to increased hospitalization for acute cardiorespiratory conditions. At present, the safety of the newer **neuraminidase** inhibitors for the treatment of influenza virus **infection** has not been established in pregnancy and they are not routinely recommended. In influenza virus **infection** complicated by pneumonia, antibacterial agents active against *Staphylococcus aureus* and *Streptococcus pneumoniae* superinfection should be used. There are few data on infective complications of asthma or COPD in pregnancy. The latter is rare, as patients with COPD are usually male and aged over 45 years. Management is the same as for nonpregnant patients. The incidence and mortality of pneumonia in pregnancy is similar to that in nonpregnant patients. Infants born to pregnant patients with pneumonia have been found to be born earlier and weigh less than controls. Risk factors for the development of pneumonia include anemia, asthma and use of antepartum corticosteroids and tocolytic agents. Based on the few available studies, the main pathogens causing pneumonia are *S. pneumoniae*, *Haemophilus influenzae*, *Mycoplasma pneumoniae* and viruses. Beta-Lactam and macrolide antibiotics therefore remain the antibiotics of choice in terms

of both pathogen coverage and safety in pregnancy. In HIV-infected pregnant patients, recurrent bacterial pneumonia, but not *Pneumocystis carinii* pneumonia (PCP), is more common than in nonpregnant patients. Trimethoprim/sulfamethoxazole (cotrimoxazole) has not definitely been associated with adverse clinical outcomes despite theoretical risks. Currently it is still the treatment of choice in PCP, where mortality remains high. In conclusion, there are few data specifically related to pregnant women with different types of LRTI. Where data are available, no significant differences compared with nonpregnant patients have been identified. In considering the use of any therapeutic agent or investigation in pregnant patients with LRTI, safety aspects must be carefully weighed against potential benefit. Otherwise, management strategies should not differ from those for nonpregnant patients. Further research in this area is warranted.

L6 ANSWER 10 OF 87 EMBASE COPYRIGHT 2005 ELSEVIER INC. ALL RIGHTS RESERVED.
on STN

ACCESSION NUMBER: 2003107935 EMBASE
TITLE: Travel epidemiology: The Saudi perspective.
AUTHOR: Memish Z.A.; Venkatesh S.; Ahmed Q.A.
CORPORATE SOURCE: Z.A. Memish, Department of Medicine, King Abdulaziz Medical City, King Fahad National Guard Hospital, P.O. Box 22490, Riyadh 11426, Saudi Arabia. memish@ngha.med.sa
SOURCE: International Journal of Antimicrobial Agents, (1 Feb 2003) 21/2 (96-101).
Refs: 38
ISSN: 0924-8579 CODEN: IAAGEA
PUBLISHER IDENT.: S 0924-8579(02)00364-3
COUNTRY: Netherlands
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 004 Microbiology
008 Neurology and Neurosurgery
017 Public Health, Social Medicine and Epidemiology
037 Drug Literature Index
048 Gastroenterology
LANGUAGE: English
SUMMARY LANGUAGE: English

AB The Kingdom of Saudi Arabia occupies four-fifths of the Arabian Peninsula, with a land area of 2 million square kilometres. Saudi Arabia holds a unique position in the Islamic world, as the custodian of the two holiest places of Islam, in Mecca and Medina. Annually, some 2 million Muslims from over 140 countries embark on Hajj. This extraordinary en masse migration is a unique forum for the study of travel epidemiology since the Hajj carries various health risks, both communicable and non-communicable, often on a colossal scale. Non-communicable hazards of the Hajj include stampede and motor vehicle trauma, fire-related burn injuries and accidental hand injury during animal slaughter. Communicable hazards in the form of outbreaks of multiple infectious **diseases** have been reported repeatedly, during and following the Hajj. Meningococcal meningitis, gastroenteritis, hepatitis A, B and C, and various zoonotic **diseases** comprise some of the possible infectious hazards at the Hajj. Many of these infectious and non-infectious hazards can be avoided or averted by adopting appropriate prophylactic measures. Physicians and health personnel must be aware of these risks to appropriately educate, immunize and prepare these travellers facing the unique epidemiological challenges of Hajj in an effort to minimize untoward effects. Travel epidemiology related to the Hajj is a new and exciting area, which offers valuable insights to the travel specialist. The sheer scale of numbers affords a rare view of migration medicine in action. As data is continually gathered and both national and international policy making is tailored to vital insights gained through travel epidemiology, the Hajj will be continually safeguarded. Practitioners will gain from findings of travel related epidemiological changes in evolution at the Hajj: the impact of vaccinating policies, **infection** control policies and

public health are afforded a real-world laboratory setting at each annual Hajj, allowing us to learn from this unique phenomenon of migration medicine. .COPYRGT. 2002 Elsevier Science B.V. and the International Society of Chemotherapy. All rights reserved.

L6 ANSWER 11 OF 87 EMBASE COPYRIGHT 2005 ELSEVIER INC. ALL RIGHTS RESERVED.
on STN

ACCESSION NUMBER: 2003258976 EMBASE
TITLE: [Role of **infection** in exacerbation and
instability of asthma].
EXACERBATION ET INSTABILITE DE L'ASTHME: QUEL ROLE JOUE L'
INFECTION?.
AUTHOR: Aubier M.; Benhamou D.; Boucot I.; Bami G.; Couderc J.-L.;
Freymuth F.; Gaillat J.; Jarlier V.; Leophonte P.; Mayaud
C.; Neukirch F.; Pappo M.; Perronne C.; Petitprez P.;
Schlemmer B.; Veyssier P.; Dusser D.; Grimfeld A.; Murris
M.; Bornstain C.; Piccoli S.
CORPORATE SOURCE: C. Mayaud, Service de Pneumologie, Hopital Tenon, 4, rue de
la Chine, 75020 Paris, France
SOURCE: Revue de Pneumologie Clinique, (2003) 59/1 (3-5).
Refs: 21
ISSN: 0761-8417 CODEN: RPCLEZ
COUNTRY: France
DOCUMENT TYPE: Journal; Editorial
FILE SEGMENT: 004 Microbiology
015 Chest Diseases, Thoracic Surgery and Tuberculosis
026 Immunology, Serology and Transplantation
037 Drug Literature Index
LANGUAGE: French

L6 ANSWER 12 OF 87 BIOTECHDS COPYRIGHT 2005 THE THOMSON CORP. on STN

ACCESSION NUMBER: 2002-08674 BIOTECHDS
TITLE: Composition useful for treatment of **mycoplasma**
infection and **diseases** associated with cell
proliferation e.g. malignancy or with co-**infection**
with another microbe, comprises agent inhibiting sialic
acid-mediated attachment of **mycoplasma**;
native or recombinant enzyme treatment and vector-mediated
gene transfer and expression in host cell for
disease therapy or prevention
AUTHOR: HIGUCHI M D L; SCHENKMAN S
PATENT ASSIGNEE: HIGUCHI M D L; SCHENKMAN S
PATENT INFO: WO 2002002050 10 Jan 2002
APPLICATION INFO: WO 2000-BR83 3 Jul 2000
PRIORITY INFO: BR 2000-2989 3 Jul 2000
DOCUMENT TYPE: Patent
LANGUAGE: English
OTHER SOURCE: WPI: 2002-154675 [20]

AB DERWENT ABSTRACT:

NOVELTY - A composition useful for treating or preventing
mycoplasma infection in a subject suffering from a
disorder characterized by increased cell proliferation or by co-
infection with a second microbe comprising an agent that prevents
or inhibits sialic acid-mediated attachment of **mycoplasma** to
the subject's cells, is new.

DETAILED DESCRIPTION - An INDEPENDENT CLAIM is also included for use
of an agent preventing/inhibiting **mycoplasma infection**
in medicaments to treat disorders characterized by increase cell
proliferation.

BIOTECHNOLOGY - Preferred Composition: The agent is preferably an
enzyme (native or recombinant) with **neuraminidase** and/or trans-
sialidase activity, especially derived from *Trypanosoma cruzi*. It
preferably has fully defined sequence (I) of 669 amino acids as given in
the specification. The medicament preferably includes a vector comprising

DNA insert of a fully defined sequence (II) of 2010 base pairs as given in the specification, producing the preferred enzyme having sequence (I) as above.

ACTIVITY - Antiatherosclerotic; antibacterial; antiviral; anti-HIV; cytostatic; vasotropic. A laboratory rat population was determined to be infected with both *Mycoplasma pulmonis* and *Chlamydia pneumoniae* using standard immunohistological techniques and histological examination of various organs. Nine adult rats (approximately 285 g; seven males, two females) presenting conjunctivitis, slow movements and, in one rat severe otitis and another severe weight loss, were allocated to groups A or B. A (Control group) comprised three animals, two of which were killed without injecting any substance and one receiving an inactivated form of 'TSC' (recombinantly produced catalytic portion of *Trypanosoma cruzi* trans-sialidase) for 5 consecutive days. B comprised five animals receiving 'TNS' (complete, native *Trypanosoma cruzi* trans-sialidase) (0.5 ml/animal, every 2 days) and sacrificed after 7, 9, and 12 days, and one rat receiving active TSC (140 microgram/day, five consecutive days) and killed after 7 days. Group B rats showed a clear improvement in symptoms, becoming more agile, requiring more ether to anesthetize them and becoming more difficult to restrain. The animal with otitis showed less equilibrium loss and the animal with severe weight loss gained weight. Since the lung was the most frequently injured organ, pulmonary alterations were examined using electron microscopy, confocal laser microscopy and immunohistochemistry. Histological sections showed that treated animals presented resolving pneumonitis after 7 d. After 9-12 days *M. pulmonis* were almost absent from alveoli and mean *C. pneumoniae* positive cell numbers in alveoli had decreased, compatible with regression of *C. pneumoniae* infection. Results are given in the specification.

MECHANISM OF ACTION - Inhibits sialic acid mediated attachment of *mycoplasma* to cells.

USE - The compositions are useful to treat diseases associated with undesirable cell proliferation, such as atherosclerotic vascular disease and malignancy (both claimed), by reducing or preventing *mycoplasma* infection. They also useful to treat diseases associated with infection with other infectious organisms co-occurring with *mycoplasma* (and typically increasing the virulence of both pathogens), especially human immunodeficiency virus or *chlamydia* species. They can be used to treat such diseases in humans or other animals, and can be administered in conjunction with conventional agents e.g. anti-platelet or chemotherapeutic agents. (63 pages)

L6 ANSWER 13 OF 87 HCAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 2002:977677 HCAPLUS
DOCUMENT NUMBER: 138:54549
TITLE: Uses of cytokines as adjuvants in avian vaccines
INVENTOR(S): Lowenthal, John William; Boyle, David Bernard; Quere, Pascale
PATENT ASSIGNEE(S): Institut National De La Recherche Agronomique, Fr.; Commonwealth Scientific and Industrial Research Organisation
SOURCE: PCT Int. Appl., 101 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002102404	A1	20021227	WO 2002-AU800	20020618
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH,				

GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR,
LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH,
PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ,
UA, UG, US, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU,
TJ, TM

RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH,
CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR,
BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.: US 2001-299047P P 20010618

AB The invention relates to a method of treatment or prophylaxis of avian pathogenic **disease** in a bird comprising administering to the bird one or more avian cytokine polypeptides sufficient to stimulate the immune response of the bird to an antigen. The avian cytokine polypeptides may be administered directly or via a nucleic acid mol. The method may further comprise administration of an antigen administered directly or via a nucleic acid mol. The invention also includes vaccines and gene constructs for carrying out the method. The vaccines and cytokines can be used to protect birds against viral and bacterial **infection** and cancer. The cytokines are selected from colony-stimulating factor, interferon, and interleukin. The birds can be poultry, domestic, or game birds.

REFERENCE COUNT: 8 THERE ARE 8 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 14 OF 87 HCAPLUS COPYRIGHT 2005 ACS on STN

ACCESSION NUMBER: 2002:676289 HCAPLUS

DOCUMENT NUMBER: 137:211942

TITLE: Drug design against drug resistant mutants using directed evolution and target protein conformation changes

INVENTOR(S): Stevens, Raymond C.; Orencia, Maria C.; Yoon, Jun S.; Hanson, Michael A.

PATENT ASSIGNEE(S): The Scripps Research Institute, USA

SOURCE: PCT Int. Appl., 82 pp.

CODEN: PIXXD2

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002068933	A2	20020906	WO 2002-US6238	20020227
WO 2002068933	A3	20021121		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN,
CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH,
GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR,
LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH,
PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ,
UA, UG, US, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU,
TJ, TM

RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH,
CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR,
BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.: US 2001-272248P P 20010228

AB The present invention provides methods for identifying new drugs and potential inhibitors and modulators of drug-resistant variants of a target protein of a drug of interest. A drug-resistant variant according to the invention has at least one mutation resulting in a structural change, an activity change or a stability change as compared to the target protein. Such variants would include natural variants such as those encountered in the clinic, but preferably variants are selected by directed evolution methodol. The present invention relates to methods for designing new drugs useful against drug-resistant bacterial cells, viruses, mammalian

cells and the like. The method involves identifying a target protein of the drug, selecting for drug-resistant variants that have an altered target protein (variant protein) by directed evolution, determining the three dimensional structure of the target and variant proteins and designing a new drug that can be effective against at least one drug-resistant variant. The present invention can be used to predict future mutations that lead to drug resistance and the type of drugs that are effective to combat such resistance.

L6 ANSWER 15 OF 87 EMBASE COPYRIGHT 2005 ELSEVIER INC. ALL RIGHTS RESERVED.
on STN

ACCESSION NUMBER: 2002046997 EMBASE
TITLE: Infectious **diseases**.
AUTHOR: Erard Ph.
CORPORATE SOURCE: Dr. Ph. Erard, Departement de Medecine, Hopital des
Cadolles, 2000 Neuchatel, Switzerland. ph.erard@net2000.ch
SOURCE: Medecine et Hygiene, (16 Jan 2002) 60/2375 (111-114).
Refs: 34
ISSN: 0025-6749 CODEN: MEHGAB
COUNTRY: Switzerland
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 004 Microbiology
015 Chest Diseases, Thoracic Surgery and Tuberculosis
037 Drug Literature Index
LANGUAGE: English
SUMMARY LANGUAGE: English

AB About 75% of antibiotic prescriptions in the outpatient setting are made for upper respiratory tract **infections**. New guidelines have been issued this year emphasizing that the vast majority of antibiotic prescriptions are not justified. More importantly, these unnecessary prescriptions are likely to contribute considerably to the emergence of antibiotic resistance. Community-acquired pneumonia is mainly caused by pneumococci and **mycoplasma**. Empirical treatment should therefore cover both groups of pathogens. Several studies have shown that **neuraminidase**-inhibitors, when administered prophylactically to family members of an index case with influenza, can prevent intrafamilial transmission of influenza. While a single dose of prophylactic doxycycline given shortly after a tick bite and removal of tick, can prevent the transmission of the Lyme agent in areas with a high (>3%) transmission rate, antibiotic treatment of patients with chronic fatigue having suffered of Lyme **disease** was of no benefit. Self-treatment of young women with acute uncomplicated cystitis has been used in clinical practice for many years. A recent prospective study validates this approach. These and other new studies should hopefully contribute to a rational and economic usage of antibiotics.

L6 ANSWER 16 OF 87 HCAPLUS COPYRIGHT 2005 ACS on STN

ACCESSION NUMBER: 2001:597831 HCAPLUS
DOCUMENT NUMBER: 135:166024
TITLE: Methods for the prevention and treatment of
infections and cancer using anti-C3b(i)
antibodies
INVENTOR(S): Taylor, Ronald P.; Lindorfer, Margaret A.; Sutherland,
William M.; Goldberg, Joanna B.
PATENT ASSIGNEE(S): The University of Virginia Patent Foundation, USA
SOURCE: PCT Int. Appl., 94 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
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WO 2001058483	A2	20010816	WO 2001-US4020	20010208
WO 2001058483	A3	20020418		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN,				
CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR,				
HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT,				
LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU,				
SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN,				
YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY,				
DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF,				
BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
CA 2400488	AA	20010816	CA 2001-2400488	20010208
EP 1257583	A2	20021120	EP 2001-907104	20010208
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,				
IE, SI, LT, LV, FI, RO, MK, CY, AL, TR				
JP 2003522159	T2	20030722	JP 2001-557591	20010208
PRIORITY APPLN. INFO.:			US 2000-181143P	P 20000208
			US 2000-724621	A 20001128
			WO 2001-US4020	W 20010208

AB The present invention relates to the treatment and prevention of viral **infections**, microbial **infections**, and septic shock by the administration of anti-C3b(i) antibodies. The present invention also relates to methods of treating and preventing viral **infection**, microbial **infection**, or septic shock in an animal comprising administering to said animal IgG antibodies, IgM antibodies and/or complement components in combination with antibodies immunospecific for C3b(i). The present invention also relates methods of treating and preventing viral **infection** or microbial **infection** in an animal comprising administering said animal antibodies that immunospecifically bind to one or more viral antigens or microbial antigens, resp., in combination with antibodies immunospecific for C3b(i). The present invention further relates methods of treating and preventing septic shock in an animal comprising administering said animal antibodies that immunospecifically bind to lipopolysaccharide, an endotoxin or a constituent of the outer wall of a gram neg. bacteria in combination with antibodies immunospecific for C3b(i). The examples discuss the use of anti-C3b(i) antibodies for the treatment and prevention of cancer.

L6 ANSWER 17 OF 87 HCAPLUS COPYRIGHT 2005 ACS on STN

ACCESSION NUMBER: 2001:792223 HCAPLUS

DOCUMENT NUMBER: 135:348878

TITLE: Therapeutic treatment and prevention of **infections** with a bioactive materials encapsulated within a biodegradable-biocompatible polymeric matrix

INVENTOR(S): Setterstrom, Jean A.; Van Hamont, John E.; Reid, Robert H.; Jacob, Elliot; Jeyanthi, Ramasubbu; Boedeker, Edgar C.; Mcqueen, Charles E.; Jarboe, Daniel L.; Cassels, Frederick; Brown, William; Thies, Curt; Tice, Thomas R.; Roberts, F. Donald; Friden, Phil

PATENT ASSIGNEE(S): United States of America as Represented by the Secretary of the Army, USA

SOURCE: U.S., 141 pp., Cont.-in-part of U.S. Ser. No. 590,973, abandoned.

CODEN: USXXAM

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 15

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 6309669	B1	20011030	US 1997-789734	19970127

US 5417986	A	19950523	US 1992-867301	19920410
US 6410056	B1	20020625	US 1995-446148	19950522
NZ 335409	A	20001222	NZ 1996-335409	19961118
US 6447796	B1	20020910	US 1997-920326	19970821
US 2003082193	A1	20030501	US 1998-13077	19980126
WO 9832427	A1	19980730	WO 1998-US1556	19980127

W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, HU, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG

AU 9863175	A1	19980818	AU 1998-63175	19980127
US 2003129233	A1	20030710	US 2002-165975	20020610
US 2003161889	A1	20030828	US 2002-224125	20020820

PRIORITY APPLN. INFO.:

US 1984-590308	B1	19840316
US 1992-867301	A2	19920410
US 1995-446148	A2	19950522
US 1995-446149	B2	19950522
US 1996-590973	B2	19960124
US 1990-493597	B2	19900315
US 1990-521945	B2	19900511
US 1991-690485	B2	19910424
US 1991-805721	B2	19911121
US 1993-34949	B1	19930322
US 1993-64559	B2	19930521
US 1994-209350	B2	19940107
US 1994-242960	A2	19940516
US 1994-247884	B2	19940523
US 1994-362944	B2	19941223
US 1996-675895	A2	19960705
US 1996-698896	A2	19960816
NZ 1996-325561	A1	19961118
US 1997-789734	A2	19970127
US 1997-920326	A1	19970821
US 1998-9986	A2	19980121
WO 1998-US1556	W	19980127

AB Novel burst-free, sustained-release biocompatible and biodegradable microcapsules which can be programmed to release their active core for variable durations ranging from 1-100 days in an aqueous physiol. environment are disclosed. The microcapsules are comprised of a core of polypeptide or other biol. active agent encapsulated in a matrix of poly(lactide/glycolide) copolymer, which may contain a pharmaceutically-acceptable adjuvant, as a blend of uncapped free carboxyl end group and end-capped forms ranging in ratios from 100/0 to 1/99. Ampicillin microcapsules effectively prevented **infection** in 73% of rats whose wound were inoculated with ampicillin-resistant strains of Staphylococcus aureus, while systemic ampicillin failed in 100% of animals.

REFERENCE COUNT: 19 THERE ARE 19 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 18 OF 87 MEDLINE on STN DUPLICATE 5
 ACCESSION NUMBER: 2001325552 MEDLINE
 DOCUMENT NUMBER: PubMed ID: 11316899
 TITLE: Autoimmune hemolytic anemia caused by IgG lambda-monotypic cold agglutinins of anti-Pr specificity after rubella **infection**.
 AUTHOR: Konig A L; Schabel A; Sugg U; Brand U; Roelcke D
 CORPORATE SOURCE: Institute of Transfusion Medicine, Katharinenhospital, Stuttgart, Germany.
 SOURCE: Transfusion, (2001 Apr) 41 (4) 488-92.

Journal code: 0417360. ISSN: 0041-1132.
PUB. COUNTRY: United States
DOCUMENT TYPE: (CASE REPORTS)
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200106
ENTRY DATE: Entered STN: 20010611
Last Updated on STN: 20010611
Entered Medline: 20010607

AB BACKGROUND: In postinfection cold agglutinin (CA) **disease**, a relation between CA specificity and the underlying infectious agent has been observed. The induction of anti-I by *Mycoplasma pneumoniae* and that of anti-i by EBV are well-established examples. CASE REPORT: A 5-year-old boy developed severe hemolytic anemia after serologically ascertained rubella **infection**. Hemolysis was caused by high-titer CAs, which were analyzed by absorption and elution with **sialidase**-treated RBCs and hemagglutination-inhibition experiments. RESULTS: After elimination of normal anti-I and anti-T, the predominant CA was found to be an IgG lambda autoantibody with anti-Pr(1) specificity. CONCLUSION: This case seems to be of interest because it is the first report of severe CA-induced hemolysis after rubella **infection**, it is the first description of an IgG lambda-monotypic CA, and, along with previous case reports (three established and three suspected cases), it indicates a relationship between rubella **infection** and the CA specificity anti-PR:

L6 ANSWER 19 OF 87 MEDLINE on STN DUPLICATE 6
ACCESSION NUMBER: 2001275243 MEDLINE
DOCUMENT NUMBER: PubMed ID: 11368254
TITLE: Vaginal microflora associated with bacterial vaginosis in nonpregnant women: reliability of **sialidase** detection.
AUTHOR: Smayevsky J; Canigia L F; Lanza A; Bianchini H
CORPORATE SOURCE: Laboratorio de Microbiologia, Centro de Educacion Medica e Investigaciones Clinicas Dr. Norberto Quirno CEMIC, Buenos Aires, Argentina.. JSmayevsky@cemic.edu.ar
SOURCE: Infectious diseases in obstetrics and gynecology, (2001) 9 (1) 17-22.
Journal code: 9318481. ISSN: 1064-7449.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200110
ENTRY DATE: Entered STN: 20011015
Last Updated on STN: 20011015
Entered Medline: 20011011

AB OBJECTIVE: To determine the prevalence of *Gardnerella vaginalis*, anaerobic bacteria and *Mycoplasma hominis* in vaginal specimens of women with and without bacterial vaginosis (BV) as well as to determine the sensitivity and specificity of the direct **sialidase** assay of vaginal fluid as a rapid test for diagnosing this syndrome. METHODS: Vaginal cultures were obtained from 109 nonpregnant women (mean age 33 +/- 7.1 years), 47 of them with clinical signs of BV (BV+) and 62 of them without BV (BV-). In addition, we determined the vaginal **sialidase** activity in both groups, which may serve as a feature of this syndrome. RESULTS: Anaerobic bacteria were isolated in 91% and 18% of the BV+ and BV- groups, respectively (p < 0.001). *Peptostreptococcus* spp., *Prevotella bivia* and *Porphyromonas* spp. were strongly associated with BV. *P. bivia* and *Prevotella* spp. represented 44% of all the anaerobes isolated in the BV+ group. All the isolated *P. bivia* strains presented **sialidase** activity. *G. vaginalis* and *M. hominis* were isolated in 76% and 42% of the BV+ and 1% and 0% of the BV- women,

respectively ($p < 0.001$). *Mobiluncus* morphotypes were observed in 34% of the BV+ and 0% of BV- women. Sensitivity, specificity, positive predictive value and negative predictive value of **sialidase** activity were 81%, 94%, 90% and 86%, respectively. CONCLUSIONS: Our data demonstrate a strong association between *G. vaginalis*, *M. hominis*, and *P. bivia* and BV. **Sialidase** activity and Gram stain of vaginal fluid represent accurate methods for diagnosing BV.

L6 ANSWER 20 OF 87 HCAPLUS COPYRIGHT 2005 ACS on STN

ACCESSION NUMBER: 2000:573950 HCAPLUS
DOCUMENT NUMBER: 133:173019
TITLE: Replication-competent porcine adenovirus-based viral vaccines
INVENTOR(S): Eloit, Marc; Klonjowski, Bernard Georges
PATENT ASSIGNEE(S): Meril, Fr.; Ecole Nationale Veterinaire De Maisons Alfort
SOURCE: PCT Int. Appl., 56 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: French
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2000047756	A1	20000817	WO 2000-FR294	20000208
W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
FR 2789695	A1	20000818	FR 1999-1813	19990211
FR 2789695	B1	20030307		
CA 2362454	AA	20000817	CA 2000-2362454	20000208
EP 1151121	A1	20011107	EP 2000-903750	20000208
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				
BR 2000008205	A	20020402	BR 2000-8205	20000208
JP 2002537766	T2	20021112	JP 2000-598651	20000208
PRIORITY APPLN. INFO.:			FR 1999-1813	A 19990211
			WO 2000-FR294	W 20000208
AB Replication competent porcine adenovirus carrying a foreign gene in the non-essential E3 region and that can be used as vaccine vectors are described. Porcine adenovirus 3 and 5 vectors are described. Construction of a number of vectors in which the E3 region is replaced is described.				
REFERENCE COUNT:		11	THERE ARE 11 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT	

L6 ANSWER 21 OF 87 EMBASE COPYRIGHT 2005 ELSEVIER INC. ALL RIGHTS RESERVED. on STN

ACCESSION NUMBER: 2001115984 EMBASE
TITLE: **Infection** and preterm labor.
AUTHOR: Yost N.P.; Cox S.M.
CORPORATE SOURCE: Dr. N.P. Yost, Department of Obstetrics, Univ. of Texas SW Medical Center, 5323 Harry Hines Boulevard, Dallas, TX 75390-9032, United States
SOURCE: Clinical Obstetrics and Gynecology, (2000) 43/4 (759-767).
Refs: 38
ISSN: 0009-9201 CODEN: COGYAK

COUNTRY: United States
DOCUMENT TYPE: Journal; General Review
FILE SEGMENT: 007 Pediatrics and Pediatric Surgery
010 Obstetrics and Gynecology
037 Drug Literature Index
LANGUAGE: English

L6 ANSWER 22 OF 87 EMBASE COPYRIGHT 2005 ELSEVIER INC. ALL RIGHTS RESERVED.
on STN DUPLICATE 7

ACCESSION NUMBER: 2000223302 EMBASE
TITLE: Coexisting anti-I/-i plus anti-Pr cold agglutinins in individual sera.
AUTHOR: Roelcke D.; Konig A.L.; Seyfert U.T.; Pereira A.
CORPORATE SOURCE: Dr. D. Roelcke, Institut fur Immunologie, Unversitat Heidelberg, Im Neuenheimer Feld 305, D-69120 Heidelberg, Germany
SOURCE: Infusionstherapie und Transfusionsmedizin, (2000) 27/3 (149-153).
Refs: 26
ISSN: 1019-8466 CODEN: IRANEE

COUNTRY: Switzerland
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 025 Hematology
LANGUAGE: English
SUMMARY LANGUAGE: English; German

AB Background: Sera with high-titer cold agglutinins (CAs) of unclear or even of apparently definite specificity may contain mixtures of CAs with different specificities. The combination of anti-I plus anti-Sia-bl CAs in sera of patients with **Mycoplasma pneumoniae infections** is well documented. No systematic studies on CA mixtures in sera of patients with other diagnoses are available. Material and Methods: Sera of 322 patients with high-titer CAs were exhaustively absorbed with **sialidase**-treated red blood cells (RBCs). By absorption, CAs against the **sialidase**-resistant I/i antigens are removed. If CAs reacting with untreated RBCs are left after absorption, they are directed against the **sialidase**- and protease-sensitive Pr1,2,3 antigens or against the **sialidase**-labile but protease-resistant antigens of the Sia-1/b/lb complex. If CA mixtures were found, specificities and isotypes of the CAs obtained by cold adsorption and warm elution were determined. Results: Three patients had mixtures of anti-i plus anti-Pr CAs, 2 patients had mixtures of anti-I plus anti-Pr CAs. Conclusion: The occurrence of CAs directed against biochemically different antigens in individual sera proves two autoimmune processes against the same cells (erythrocytes) in the same patient. One explanation for this constellation would be a postinfection cold agglutination in a patient with chronic CA disease.

L6 ANSWER 23 OF 87 HCAPLUS COPYRIGHT 2005 ACS on STN

ACCESSION NUMBER: 1999:659405 HCAPLUS
DOCUMENT NUMBER: 131:285411
TITLE: Avian IL-15 nucleotides and polypeptides, and methods of immunizing poultry using avian IL-15
INVENTOR(S): Choi, Kang; Tsusaki, Yoshinari; Kamogawa, Koichi; Lillehoj, Hyun S.
PATENT ASSIGNEE(S): Nippon Zeon Co., Ltd., Japan; United States Dept. of Agriculture
SOURCE: PCT Int. Appl., 66 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
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WO 9951622	A1	19991014	WO 1999-US7485	19990406
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SL, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
AU 9934720	A1	19991025	AU 1999-34720	19990406
JP 11346786	A2	19991221	JP 1999-98329	19990406
PRIORITY APPLN. INFO.:			US 1998-55293	A 19980406
			WO 1999-US7485	W 19990406

AB The present invention relates to an isolated avian IL-15 polypeptide comprising: (a) the amino acid sequence of SEQ ID NO:1; (b) fragments of the amino acid sequence of SEQ ID NO:1, wherein said fragments stimulate growth of avian T lymphocytes expressing $\gamma\delta$ TCR; or (c) the amino acid sequence of SEQ ID NO:1 having one or more amino acid substitutions, mutations, deletions and insertions and to polynucleotides encoding the amino acid sequences. The present invention further encompasses methods of recombinantly producing said amino acid and polynucleotide sequences and methods of using the amino acid and polynucleotide sequences, particularly for avian vaccines. The sequence of chicken IL-15, SEQ ID Nos:1 and 2 are described. Thus, recombinant fowlpox virus fNZ29R/IL-15 was constructed and purified, and expression of fNZ29R/IL-15 was verified.

REFERENCE COUNT: 3 THERE ARE 3 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 24 OF 87 HCAPLUS COPYRIGHT 2005 ACS on STN

ACCESSION NUMBER: 1999:344861 HCAPLUS
DOCUMENT NUMBER: 131:4240
TITLE: Immunoglobulin molecules having a synthetic variable region and modified specificity
INVENTOR(S): Burch, Ronald M.
PATENT ASSIGNEE(S): Euro-Celtique, S.A., Bermuda
SOURCE: PCT Int. Appl., 123 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9925378	A1	19990527	WO 1998-US24302	19981113
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
CA 2309990	AA	19990527	CA 1998-2309990	19981113
CA 2310269	AA	19990527	CA 1998-2310269	19981113
WO 9925379	A1	19990527	WO 1998-US24303	19981113
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				

RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES,
FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI,
CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

AU 9914597	A1	19990607	AU 1999-14597	19981113
AU 763029	B2	20030710		
AU 9914598	A1	19990607	AU 1999-14598	19981113
AU 737457	B2	20010823		
EP 1030684	A1	20000830	EP 1998-958584	19981113
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				
EP 1032420	A1	20000906	EP 1998-958583	19981113
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				
JP 2001526021	T2	20011218	JP 2000-520811	19981113
BR 9815289	A	20011226	BR 1998-15289	19981113
BR 9815580	A	20020129	BR 1998-15580	19981113
JP 2002507544	T2	20020312	JP 2000-520812	19981113
ZA 9900048	A	19990708	ZA 1999-48	19990105
ZA 9900049	A	20000309	ZA 1999-49	19990105
US 2002028469	A1	20020307	US 2001-963232	20010926
BR 2002012865	A	20040914	BR 2002-12865	20020828

PRIORITY APPLN. INFO.:

US 1997-65716P	P	19971114
US 1998-81403P	P	19980410
US 1998-191780	A1	19981113
WO 1998-US24302	W	19981113
WO 1998-US24303	W	19981113
US 2001-963232	A	20010926
WO 2002-US27446	W	20020828

AB The invention provides modified Ig mols., particularly antibodies, that immunospecifically bind a first member of a binding pair which binding pair consists of the first member and a second member, which Igs have a variable domain containing one or more complimentary determining regions that contain the amino acid sequence of a binding site for the second member of the binding pair. The first member is a tumor antigen or an antigen of an infectious disease agent, and the second member is a mol. on the surface of an immune cell. The invention further provides for therapeutic and diagnostic use of the modified Ig.

REFERENCE COUNT: 13 THERE ARE 13 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 25 OF 87 HCAPLUS COPYRIGHT 2005 ACS on STN

ACCESSION NUMBER: 1999:467965 HCAPLUS
DOCUMENT NUMBER: 131:115304
TITLE: Recombinant herpesvirus of turkeys and uses thereof
INVENTOR(S): Cochran, Mark D.
PATENT ASSIGNEE(S): Syntro Corp., USA
SOURCE: U.S., 92 pp.
CODEN: USXXAM
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 18
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
-----	----	-----	-----	-----
US 5928648	A	19990727	US 1993-23610	19930226
US 4877737	A	19891031	US 1985-773430	19850906
US 5068192	A	19911126	US 1986-823102	19860127
WO 8701287	A1	19870312	WO 1986-US1804	19860903
W: AU, DK, JP				
RW: AT, BE, CH, DE, FR, GB, IT, LU, NL, SE				
AU 8663717	A1	19870324	AU 1986-63717	19860903
EP 237546	A1	19870923	EP 1986-905609	19860903
R: AT, BE, CH, DE, FR, GB, IT, LI, LU, NL, SE				

JP 63501122	T2	19880428	JP 1986-504917	19860903
EP 256092	A1	19880224	EP 1987-901222	19870123
EP 256092	B1	19980408		
R: AT, BE, CH, DE, FR, GB, IT, LI, LU, NL, SE				
EP 794257	A1	19970910	EP 1997-103457	19870123
R: AT, BE, CH, DE, FR, GB, IT, LI, LU, NL, SE				
AT 164885	E	19980415	AT 1987-901222	19870123
CA 1339468	A1	19970923	CA 1987-528117	19870126
IL 81398	A1	19970814	IL 1987-81398	19870127
FR 2601689	A1	19880122	FR 1987-10142	19870717
FR 2601689	B1	19921016		
US 5047237	A	19910910	US 1988-192866	19880511
US 5223424	A	19930629	US 1988-225032	19880727
EP 658623	A2	19950621	EP 1995-100565	19880727
EP 658623	A3	19950927		
R: BE, DE, FR, GB, IT, NL				
AU 9210266	A1	19920514	AU 1992-10266	19920115
AU 656553	B2	19950209		
WO 9325665	A1	19931223	WO 1993-US5681	19930614
W: AU, CA, HU, JP, KR, PL, RO, RU				
RW: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
AU 9345362	A1	19940104	AU 1993-45362	19930614
AU 684046	B2	19971204		
EP 644931	A1	19950329	EP 1993-915344	19930614
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LI, LU, MC, NL, PT, SE				
JP 08500969	T2	19960206	JP 1993-501778	19930614
US 5506128	A	19960409	US 1993-78873	19930617
US 5593873	A	19970114	US 1994-247475	19940523
US 5961982	A	19991005	US 1994-288065	19940809
US 5731188	A	19980324	US 1994-323531	19941014
US 5965138	A	19991012	US 1994-362240	19941222
US 5763269	A	19980609	US 1995-384476	19950201
US 5599544	A	19970204	US 1995-479650	19950607
US 5853733	A	19981229	US 1996-663566	19960613
US 5804372	A	19980908	US 1996-674169	19960701
US 6121043	A	20000919	US 1997-915520	19970815
US 6210961	B1	20010403	US 1997-912803	19970818
US 2002081316	A1	20020627	US 2001-881457	20010614
PRIORITY APPLN. INFO.:				
			US 1985-773403	A2 19850906
			US 1985-773430	YY 19850906
			US 1985-773430	A2 19850906
			US 1986-823102	YY 19860127
			US 1986-823102	A2 19860127
			US 1986-823102	YY 19860127
			US 1986-887140	B2 19860717
			US 1986-902877	B2 19860902
			US 1986-902887	B2 19860902
			US 1986-933107	B2 19861120
			US 1987-78519	B2 19870727
			US 1988-225032	A2 19880727
			US 1991-649380	B2 19910131
			US 1991-696262	B2 19910430
			US 1992-898087	B2 19920612
			US 1992-914057	B2 19920713
			WO 1986-US1804	A 19860903
			EP 1987-901222	A3 19870123
			US 1988-192866	A2 19880519
			EP 1988-907889	A3 19880727
			US 1991-732584	B1 19910718
			US 1992-926784	B1 19920807
			US 1993-23610	A 19930226
			US 1993-37707	B1 19930325
			WO 1993-US5681	A 19930614
			US 1993-117633	B1 19930907

US 1994-247475	A1 19940523
US 1994-288065	A1 19940809
US 1994-334428	A1 19941104
WO 1995-US10245	A2 19950809
US 1996-663566	A2 19960613
US 1997-804372	A1 19970221
US 1999-426352	B2 19991025

AB The present invention relates to a recombinant herpesvirus of turkeys comprises foreign DNA inserted into a site in the herpesvirus of turkeys genome which is not essential for replication of the herpesvirus of turkeys. The invention further relates to homol. vectors which produce recombinant herpesvirus of turkeys by inserting foreign DNA into herpesvirus of turkeys genome. Genetically-engineered virus S-FPV-062 is described in the Materials and Methods section which follows. One advantage of recombinant HVT as live vaccines is that they may be engineered to express only a limited number of antigens that are needed to confer protective immunity to the corresponding pathogens. Consequently, host animals vaccinated with the recombinant HVT can be distinguished from those which have been infected with the wild type virus by the absence of antigens that are normally present in the wild type virus.

REFERENCE COUNT: 31 THERE ARE 31 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 26 OF 87 MEDLINE on STN DUPLICATE 8
 ACCESSION NUMBER: 1999344855 MEDLINE
 DOCUMENT NUMBER: PubMed ID: 10416366
 TITLE: Interactions of **Mycoplasma** bovoculi with erythrocytes: role of p94 surface protein.
 AUTHOR: Salih B A; Rosenbusch R F
 CORPORATE SOURCE: Department of Microbiology, Immunology and Preventive Medicine, Iowa State University, Ames, USA.
 SOURCE: Zentralblatt fur Veterinarmedizin. Reihe B. Journal of veterinary medicine. Series B, (1999 Jun) 46 (5) 323-9. Journal code: 0331325. ISSN: 0514-7166.
 PUB. COUNTRY: GERMANY: Germany, Federal Republic of
 DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
 LANGUAGE: English
 FILE SEGMENT: Priority Journals
 ENTRY MONTH: 199909
 ENTRY DATE: Entered STN: 19990913
 Last Updated on STN: 20030218
 Entered Medline: 19990902

AB The attachment of two strains of **Mycoplasma** bovoculi to erythrocytes was measured using 35S-methionine-labelled organisms. Receptor sites of M. bovoculi involved in this attachment are trypsin-sensitive, since mild trypsin treatment of the intact organisms abolished this process completely. Pretreatment of erythrocytes with trypsin or increasing concentrations of **neuraminidase** resulted in no measurable effect. Monoclonal antibody MA25.5 directed against a M. bovoculi surface antigen of 94 kDa termed p94 blocked 40% of the attachment, while MA18.13 directed against a 57 kDa protein band of M. bovoculi had no effect on the attachment process. Other properties of M. bovoculi were tested using six strains of the **mycoplasma** and erythrocytes from several animal species. None of the strains showed haemagglutinating or haemadsorbing activities.

L6 ANSWER 27 OF 87 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation. on STN
 ACCESSION NUMBER: 1999:345454 BIOSIS
 DOCUMENT NUMBER: PREV199900345454
 TITLE: Utility of the gram stain and the **sialidase** detection for diagnosis of bacterial vaginosis (BV) in non-pregnant women (NPW).
 AUTHOR(S): Smayevsky, J. [Reprint author]; Roldan, L.; Fernandez

CORPORATE SOURCE: Canigia, L.; Lanza, A.; Bianchini, H.; Decca, L.
 SOURCE: CEMIC, Buenos Aires, Argentina
 Abstracts of the General Meeting of the American Society
 for Microbiology, (1999) Vol. 99, pp. 182. print.
 Meeting Info.: 99th General Meeting of the American Society
 for Microbiology. Chicago, Illinois, USA. May 30-June 3,
 1999. American Society for Microbiology.
 ISSN: 1060-2011.
 DOCUMENT TYPE: Conference; (Meeting)
 Conference; Abstract; (Meeting Abstract)
 Conference; (Meeting Poster)
 LANGUAGE: English
 ENTRY DATE: Entered STN: 24 Aug 1999
 Last Updated on STN: 24 Aug 1999

L6 ANSWER 28 OF 87 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation. on
 STN

ACCESSION NUMBER: 2000:139503 BIOSIS
 DOCUMENT NUMBER: PREV200000139503
 TITLE: Utility of the Gram stain and the **sialidase**
 detection for diagnosis of bacterial vaginosis (BV) in
 non-pregnant women (NPW).
 AUTHOR(S): Smayevsky, J. [Reprint author]; Roldan, L.; Fernandez
 Canigia, L. [Reprint author]; Lanza, A. [Reprint author];
 Arganara, M.; Bianchini, H. [Reprint author]; Decca, L.
 CORPORATE SOURCE: Laboratorio de Microbiologia de CEMIC, Buenos Aires,
 Argentina
 SOURCE: International Journal of Gynecology and Obstetrics, (Nov.,
 1999) Vol. 67, No. Suppl. 1, pp. S51. print.
 Meeting Info.: Second International Meeting on Bacterial
 Vaginitis. Aspen, Colorado, USA. September 17-19, 1998.
 CODEN: IJGOAL. ISSN: 0020-7292.
 DOCUMENT TYPE: Conference; (Meeting)
 Conference; Abstract; (Meeting Abstract)
 LANGUAGE: English
 ENTRY DATE: Entered STN: 19 Apr 2000
 Last Updated on STN: 4 Jan 2002

L6 ANSWER 29 OF 87 HCAPLUS COPYRIGHT 2005 ACS on STN

ACCESSION NUMBER: 1998:527193 HCAPLUS
 DOCUMENT NUMBER: 129:166193
 TITLE: Therapeutic treatment and prevention of
infections with a bioactive material
 encapsulated within a biodegradable-biocompatible
 polymeric matrix
 INVENTOR(S): Setterstrom, Jean A.; Van Hamont, John E.; Reid,
 Robert H.; Jacob, Elliot; Jeyanthi, Ramasubbu;
 Boedeker, Edgar C.; McQueen, Charles E.; Tice, Thomas
 R.; Roberts, F. Donald; Friden, Phil
 PATENT ASSIGNEE(S): United States Dept. of the Army, USA; Van Hamont, John
 E.; et al.
 SOURCE: PCT Int. Appl., 363 pp.
 CODEN: PIXXD2
 DOCUMENT TYPE: Patent
 LANGUAGE: English
 FAMILY ACC. NUM. COUNT: 15
 PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9832427	A1	19980730	WO 1998-US1556	19980127
W:	AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE,			
	DK, EE, ES, FI, GB, GE, GH, HU, IL, IS, JP, KE, KG, KP, KR, KZ,			
	LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL,			

PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US,
 UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
 RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, DE, DK, ES, FI,
 FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM,
 GA, GN, ML, MR, NE, SN, TD, TG

US 6309669 B1 20011030 US 1997-789734 19970127
 AU 9863175 A1 19980818 AU 1998-63175 19980127

PRIORITY APPLN. INFO.: US 1997-789734 A 19970127
 US 1984-590308 B1 19840316
 US 1992-867301 A2 19920410
 US 1995-446148 A2 19950522
 US 1995-446149 B2 19950522
 US 1996-590973 B2 19960124
 WO 1998-US1556 W 19980127

AB Novel burst-free, sustained release biocompatible and biodegradable microcapsules are disclosed which can be programmed to release their active core for variable durations ranging from 1-100 days in an aqueous physiol. environment. The microcapsules are comprised of a core of polypeptide or other biol. active agent encapsulated in a matrix of poly(lactide/glycolide) copolymer, which may contain a pharmaceutically acceptable adjuvant, as a blend of uncapped free carboxyl end group and end-capped forms ranging in ratios from 100/0 to 1/99.

REFERENCE COUNT: 6 THERE ARE 6 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 30 OF 87 MEDLINE on STN DUPLICATE 9

ACCESSION NUMBER: 1998274733 MEDLINE

DOCUMENT NUMBER: PubMed ID: 9611799

TITLE: Identification of two glycosylated components of **Mycoplasma penetrans**: a surface-exposed capsular polysaccharide and a glycolipid fraction.

AUTHOR: Neyrolles O; Brenner C; Prevost M C; Fontaine T; Montagnier L; Blanchard A

CORPORATE SOURCE: Unite d'Oncologie Virale, Institut Pasteur, Paris, France.

SOURCE: Microbiology (Reading, England), (1998 May) 144 (Pt 5) 1247-55.

Journal code: 9430468. ISSN: 1350-0872.

PUB. COUNTRY: ENGLAND: United Kingdom

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English

FILE SEGMENT: Priority Journals

ENTRY MONTH: 199807

ENTRY DATE: Entered STN: 19980716

Last Updated on STN: 19980716

Entered Medline: 19980707

AB Among the wall-less **mycoplasmas** only a few species have been identified with a capsule at their cell surface. **Mycoplasma penetrans** is a recently identified **mycoplasma** with unique morphology, isolated from HIV-infected patients. Using transmission electron microscopy, it was found that M. penetrans is surrounded by capsular material 11 nm (strain GTU-54-6A1) to 30 nm (strain HF-2) thick, which can be stained with ruthenium red and labelled with cationized ferritin. The polysaccharide composition of this capsule was indicated by its staining with periodic acid-thiocarbohydrazide silver proteinate and the abolition of ruthenium red staining of the cell surface by **neuraminidase** treatment. In addition, proteinase K treatment of the M. penetrans cells resulted in removal of the capsule, suggesting that polypeptides may contribute in anchoring it to the membrane or in its stability. Two different types of glycosylated material were detected in **mycoplasma** extracts by SDS-PAGE and periodic acid-Schiff staining. The first component was a high-molecular-mass material, which was heat- and proteinase-K-labile and which probably constitutes the capsular polymer. The other component was a low-molecular-mass glycolipid fraction, which was proteinase-K-, heat- and EDTA-resistant. The

identification of a capsule at the *M. penetrans* cell surface is of particular interest for a **mycoplasma** which has been shown to adhere to various host cells and to penetrate into their intracellular compartments. The capsule may have significance in the pathogenesis of **disease** associated with **infection** by this organism.

L6 ANSWER 31 OF 87 MEDLINE on STN DUPLICATE 10
ACCESSION NUMBER: 1998354165 MEDLINE
DOCUMENT NUMBER: PubMed ID: 9689740
TITLE: *Pasteurella haemolytica* complicated respiratory **infections** in sheep and goats.
AUTHOR: Brogden K A; Lehmkuhl H D; Cutlip R C
CORPORATE SOURCE: Respiratory and Neurologic Disease Research Unit, National Animal Disease Center, Agricultural Research Service, U.S. Department of Agriculture, Ames, IA 50010, USA.
SOURCE: Veterinary research, (1998 May-Aug) 29 (3-4) 233-54. Ref: 156
Journal code: 9309551. ISSN: 0928-4249.
PUB. COUNTRY: France
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199809
ENTRY DATE: Entered STN: 19980917
Last Updated on STN: 19980917
Entered Medline: 19980908
AB Respiratory **infections** which commonly occur in sheep and goats often result from adverse physical and physiological stress combined with viral and bacterial **infections**. Inevitably, *Pasteurella haemolytica* pneumonia occurs as a result of these interactions. In this review, we present recent advances in research on the complex etiology of pneumonia involving *P. haemolytica*. Initially stress, induced by factors such as heat, overcrowding, exposure to inclement weather, poor ventilation, handling and transport is a major predisposing factor. Respiratory viruses including parainfluenza 3 (PI-3) virus, adenovirus type 6 and respiratory syncytial virus (RSV), and to a lesser extent bovine adenovirus type 2, ovine adenovirus types 1 and 5, and reovirus type 1 cause respiratory **infections** and pneumonia. More importantly these viruses also dramatically increase the susceptibility of sheep and goats to secondary *P. haemolytica* **infection**. Primary **infection** of the lower respiratory tract, with **Mycoplasma** ovipneumoniae and *Bordetella parapertussis* can increase the susceptibility of sheep and goats to secondary *P. haemolytica* **infection**. It is possible that initial **infections** with viral or primary bacterial agents break down the antimicrobial barrier consisting of beta defensins and anionic peptides found in epithelial cells, resident and inflammatory cells, and serous and mucous secretions of the respiratory tract. Loss of barrier integrity may release *P. haemolytica* from its usual commensal status. Once in the lung, *P. haemolytica* becomes opportunistic. To grow and colonize, *P. haemolytica* uses extracellular products like O-sialoglycoprotein endopeptidase, **neuraminidase** and RTX leukotoxin, as well as cell-associated products such as capsular polysaccharide, lipopolysaccharide, outer membrane proteins, proteins involved in iron acquisition and a periplasmic superoxide dismutase. In lambs and kids, pneumonic pasteurellosis can be acute, characterized by fever, listlessness, poor appetite and sudden death. Sheep and goats that survive the acute stage may recover or become chronically affected showing reduced lung capacity and weight gain efficiency and sporadic deaths may occur. This **infection** is detrimental to sheep and goats throughout the world and flocks and herds of small ranches, dairy operations, or large feedlots are all affected.

L6 ANSWER 32 OF 87 MEDLINE on STN
 ACCESSION NUMBER: 1998019503 MEDLINE
 DOCUMENT NUMBER: PubMed ID: 9356671
 TITLE: CMV-induced anti-Sia-b1 cold agglutinin in an immunocompromised patient.
 AUTHOR: Zilow G; Haffner D; Roelcke D
 CORPORATE SOURCE: Institut fur Immunologie, Ruprecht-Karls-Universitat Heidelberg, Germany.
 SOURCE: Beitrage zur Infusionstherapie und Transfusionsmedizin = Contributions to infusion therapy and transfusion medicine, (1997) 34 180-4.
 Journal code: 9442459. ISSN: 1023-2028.
 PUB. COUNTRY: Switzerland
 DOCUMENT TYPE: (CASE REPORTS)
 Journal; Article; (JOURNAL ARTICLE)
 LANGUAGE: English
 FILE SEGMENT: Priority Journals
 ENTRY MONTH: 199712
 ENTRY DATE: Entered STN: 19980116
 Last Updated on STN: 19980116
 Entered Medline: 19971230

AB In postinfection cold agglutination, certain cold agglutinin (CA) specificities are associated with distinct infectious agents. The combined occurrence of anti-I and anti-Sia-b1 CAs following **Mycoplasma pneumoniae infection** has been reported recently. After renal transplantation and hyperacute graft rejection, transiently occurring CAs were observed in an 18-year-old boy. The CAs were characterized by serum cold absorption with **sialidase** -treated red cells and warm elution from the cells. An anti-Sia-b1 CA could be differentiated from an accompanying low-liter anti-I. Fresh **infections** with **Mycoplasma pneumoniae**, Epstein-Barr virus, rubella, and varicella viruses were excluded, but CMV **infection** was demonstrated. This is the first case of a postinfection anti-Sia-b1 CA associated with CMV **infection**.

L6 ANSWER 33 OF 87 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation. on STN
 ACCESSION NUMBER: 1996:139420 BIOSIS
 DOCUMENT NUMBER: PREV199698711555
 TITLE: Vaginal and cervical fluid **sialidase** activity associated with cervicovaginal microorganisms and preterm labor.
 AUTHOR(S): Pennacchi, L. [Reprint author]; Coata, G. [Reprint author]; De Domenico, P. [Reprint author]; Sensini, A.; Marangi, M.; Tassi, C.; Di Renzo, G. C. [Reprint author]
 CORPORATE SOURCE: Inst. Ob/Gyn, Univ. Perugia, Perugia, Italy
 SOURCE: American Journal of Obstetrics and Gynecology, (1996) Vol. 174, No. 1 PART 2, pp. 400.
 Meeting Info.: 16th Annual Meeting of the Society of Perinatal Obstetricians. Kamuela, Hawaii, USA. February 4-10, 1996.
 CODEN: AJOGAH. ISSN: 0002-9378.
 DOCUMENT TYPE: Conference; (Meeting)
 Conference; Abstract; (Meeting Abstract)
 Conference; (Meeting Poster)
 LANGUAGE: English
 ENTRY DATE: Entered STN: 3 Apr 1996
 Last Updated on STN: 26 Apr 1996

L6 ANSWER 34 OF 87 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation. on STN
 ACCESSION NUMBER: 1996:422632 BIOSIS
 DOCUMENT NUMBER: PREV199699153688
 TITLE: Mechanisms and factors involved in **Mycoplasma**

bovis adhesion to host cells.
AUTHOR(S): Sachse, Konrad [Reprint author]; Grajetzki, Christine;
Rosengarten, Renate; Haenel, Ingrid; Heller, Martin;
Pfuetzner, Horst
CORPORATE SOURCE: Bundesinst. Gesundheitlichen Verbraucherschutz und
Veterinaermed., Fachbereich 4, Naumburger Str. 96a, D-07743
Jena, Germany
SOURCE: Zentralblatt fuer Bakteriologie, (1996) Vol. 284, No. 1,
pp. 80-92.
CODEN: ZEBAE8. ISSN: 0934-8840.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 26 Sep 1996
Last Updated on STN: 26 Sep 1996

AB **Mycoplasma** (M.) bovis cytheadhesion was studied using permanent embryonic bovine lung (EBL) cells as host system. Adherence rates were found to be strongly dependent on temperature and the **mycoplasma** -to-EBL ratio near the point of saturation of the attachment isotherm was determined to be 225:1. Mild trypsinization of viable M. bovis cells caused a measurable decrease of adherence indicating that surface proteins, among them the P26 antigen, played a major part as adhesion factors. **Neuraminidase** treatment of **mycoplasmas** led to a drastic reduction of adherence rates, which emphasizes the importance of sialyl moieties in adhesive interactions. The ability of the P26 antigen, a hydrophilic 32-kDa protein, to function as an adhesin was confirmed using a competitive adherence assay, in which the HPLC-purified protein was shown to reduce **mycoplasma** adhesion. These data complement previous findings obtained with the corresponding monoclonal antibody (MAb) 4F6. In further inhibition experiments, it could be demonstrated that MAb 1E5, which is directed against a common epitope of at least three members of the Vsp (variable surface protein) family of M. bovis, was also capable of decreasing **mycoplasma** attachment to EBL cells. This is the first evidence of possible involvement of Vsps in cytheadhesion. In an effort to identify more putative adhesion proteins of this organism, the reverse adherence screening assay was used, a procedure based on the specific binding of labelled mammalian tissue culture cells to Western-blotted mycoplasmal proteins.

L6 ANSWER 35 OF 87 BIOTECHDS COPYRIGHT 2005 THE THOMSON CORP. on STN
DUPLICATE 12

ACCESSION NUMBER: 1995-00162 BIOTECHDS

TITLE: Recombinant avipox virus combining DNA which encodes a polypeptide exhibiting antigenicity of **Mycoplasma gallisepticum**;
e.g. varicella-zoster virus vector containing a gene encoding a fusion protein of an antigen with a Newcastle-disease virus anchor peptide for use as a recombinant vaccine

AUTHOR: Saito S; Ohkawa S; Saeki S; Ohsawa I; Funato H; Iritani Y;
Aoyama S; Takahashi K; Asamura K

PATENT ASSIGNEE: Japan-Zeon; Shionogi

PATENT INFO: WO 9423019 13 Oct 1994

APPLICATION INFO: WO 1994-JP541 31 Mar 1994

PRIORITY INFO: JP 1993-245625 30 Sep 1993; JP 1993-74139 31 Mar 1993

DOCUMENT TYPE: Patent

LANGUAGE: Japanese

OTHER SOURCE: WPI: 1994-333181 [41]

AB A new antigenic protein capable of reacting with **Mycoplasma gallisepticum** immune serum or infected serum is encoded by an M. gallisepticum gene with a specified restriction map, or is a variant with equivalent antigenicity. The antigen may be produced as a fusion protein with a signal membrane anchor of a type-II outer membrane protein of a bird virus at the 5'-terminus, using an avipox virus vector. The anchor sequence may be from a Newcastle-disease virus hemagglutinin-

neuraminidase gene. The DNA sequence encoding the fusion protein is specified. The recombinant avipox virus, recombinant antigen and fusion protein are useful in production of a live recombinant vaccine against *M. gallisepticum* infection. In an example, a live vaccine strain of varicella-zoster virus NP was used to infect a CEF cell culture monolayer at an MOI of 0.1, and after 3 hr cells were electroporated with plasmid pNZ7929-R1 (3.0 kV/cm, 0.4 msec and 25 deg). Cells with the plasmid were cultured for 72 hr at 37 deg, freeze-thawed and the recombinant virus was collected (fNZ7929-R1). (123pp)

L6 ANSWER 36 OF 87 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation. on STN

ACCESSION NUMBER: 1994:547464 BIOSIS
DOCUMENT NUMBER: PREV199598007012
TITLE: Adherence of *Ureaplasma urealyticum* to human epithelial cells.
AUTHOR(S): Smith, D. G. E.; Russell, W. C.; Thirkell, D. [Reprint author]
CORPORATE SOURCE: Div. Cell Molecular Biol., Sch. Biological Med. Sci., Univ. St. Andrews, Irvine Build., North St., St. Andrews, Fife KY16 9AL, UK
SOURCE: Microbiology (Reading), (1994) Vol. 140, No. 10, pp. 2893-2898.
ISSN: 1350-0872.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 22 Dec 1994
Last Updated on STN: 22 Dec 1994

AB Adherence of *Ureaplasma urealyticum* cells to eukaryotic cell monolayers was quantified using the Bertholet assay to monitor ammonia produced from urea by ureaplasma urease. Adherence was abolished by pre-treatment of ureaplasmas with HeLa cell extracts and inhibited to varying degrees by pretreatment of the ureaplasmas with N-acetylneuraminic acid, specific antisera and monoclonal antibodies. The data suggest the presence of several ureaplasma adhesins, some of which are species- or serotype-specific and some of which are proteinaceous and antigenic. The serotype-8-specific 96 kDa surface-expressed antigen may be one adhesin. Pre-treatment of HeLa cell monolayers with **neuraminidase** significantly reduced ureaplasma adherence and, using a novel 'immunoblot adherence assay', ureaplasmas were shown to bind to a number of HeLa cell components, three of which appear to terminate in sialic acid.

L6 ANSWER 37 OF 87 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation. on STN DUPLICATE 13

ACCESSION NUMBER: 1994:272691 BIOSIS
DOCUMENT NUMBER: PREV199497285691
TITLE: Microtiter plate adherence assay and receptor analogs for **Mycoplasma hyopneumoniae**.
AUTHOR(S): Zhang, Qijing; Young, Theresa F.; Ross, Richard F. [Reprint author]
CORPORATE SOURCE: Vet. Med. Research Inst., Iowa State Univ., 1802 Elwood Dr., Ames, IA 50011, USA
SOURCE: Infection and Immunity, (1994) Vol. 62, No. 5, pp. 1616-1622.
CODEN: INFIBR. ISSN: 0019-9567.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 24 Jun 1994
Last Updated on STN: 25 Jun 1994

AB A microliter plate adherence assay for **Mycoplasma hyopneumoniae** was established by use of purified swine tracheal cilia which contained receptors for the **mycoplasmas**. *M. hyopneumoniae* bound specifically to plates coated with solubilized cilia. The binding was dependent on both the concentration of cilia and the number of

mycoplasmas. Dextran sulfate, heparin, chondroitin. sulfate, laminin, mucin, and fucoidan significantly inhibited the binding of the **mycoplasmas**. The six inhibitors also disrupted the adherence of the **mycoplasmas** to intact ciliated cells. Preincubation with either **mycoplasmas** or cilia indicated that heparin, mucin, fucoidan, and chondroitin sulfate interacted with the adhesive molecules on the surface of the **mycoplasmas**, while laminin blocked the receptors in cilia. The basis for the inhibition induced by dextran sulfate was unknown. Treatment of cilia with **neuraminidase** appeared to promote adherence of the **mycoplasmas**, whereas treatment of cilia with sodium metaperiodate decreased binding. These results indicate that receptors for *M. hyopneumoniae* in the ciliated epithelium of the respiratory tract of pigs are glycoconjugate in nature.

L6 ANSWER 38 OF 87 MEDLINE on STN DUPLICATE 14
 ACCESSION NUMBER: 94219513 MEDLINE
 DOCUMENT NUMBER: PubMed ID: 8166188
 TITLE: Bacterial vaginosis is associated with prematurity and vaginal fluid mucinase and **sialidase**: results of a controlled trial of topical clindamycin cream.
 AUTHOR: McGregor J A; French J I; Jones W; Milligan K; McKinney P J; Patterson E; Parker R
 CORPORATE SOURCE: Department of Obstetrics and Gynecology, University of Colorado Health Sciences Center, Denver 80262.
 SOURCE: American journal of obstetrics and gynecology, (1994 Apr) 170 (4) 1048-59; discussion 1059-60.
 Journal code: 0370476. ISSN: 0002-9378.
 PUB. COUNTRY: United States
 DOCUMENT TYPE: (CLINICAL TRIAL)
 Journal; Article; (JOURNAL ARTICLE)
 (MULTICENTER STUDY)
 (RANDOMIZED CONTROLLED TRIAL)
 LANGUAGE: English
 FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
 ENTRY MONTH: 199405
 ENTRY DATE: Entered STN: 19940606
 Last Updated on STN: 19940606
 Entered Medline: 19940526
 AB OBJECTIVE: The pathogenesis of preterm birth and other adverse pregnancy outcomes linked with reproductive tract **infection** remains poorly understood. Mucolytic enzymes, including mucinases and **sialidases** (**neuraminidase**), are recognized virulence factors among enteropathogens and bacteria that cause periodontal **infection**. Perturbation of maternal cervicovaginal mucosa membrane host defenses by such enzyme-producing microorganisms may increase the risk of subclinical intrauterine **infection** during pregnancy and thus increase risks of preterm birth. STUDY DESIGN: We prospectively evaluated vaginal fluid mucinase and **sialidase** and selected cervicovaginal bacteria along with pregnancy outcomes in 271 women. Within this study, women with bacterial vaginosis (16 to 27 week' gestation) were treated with 2% clindamycin vaginal cream or placebo. Enzyme, microbial findings, treatment effects, and pregnancy outcomes were compared among drug- and placebo-treated women and control women without bacterial vaginosis. RESULTS: Presence of bacterial vaginosis at intake was associated with increased risk of preterm birth (relative risk 3.3, 95% confidence interval 1.2 to 9.1, p = 0.02), premature rupture of membranes (relative risk 3.8, 95% confidence interval 1.6 to 9.0, p = 0.002), and preterm premature rupture of membranes. Mucinase and **sialidase** activities were more commonly identified, and they occurred in higher concentrations, if present, in women with bacterial vaginosis (mucinase: 44.3% with bacterial vaginosis vs 27.4% without, p = 0.007; **sialidase**: 45% with bacterial vaginosis vs 12% without p < 0.001). **Sialidase** activity was associated with bacterial vaginosis-linked organisms (*Gardnerella vaginalis*, *Mobiluncus* spp, and *Mycoplasma*

hominis) and Chlamydia trachomatis and yeast species; mucinase activity was associated only with bacterial vaginosis-linked microorganisms. Clindamycin, 2% cream, was effective treatment for bacterial vaginosis and temporarily reduced mucinase and sialidase activities. Topical treatment of bacterial vaginosis did not reduce risks of perinatal morbidity. Women with persistent or recurrent sialidase 8 weeks after treatment were at increased risk of preterm birth (15.6% vs 7.4%) premature rupture of membranes (30% vs 15%), and low birth weight (20% vs 3%, relative risk 6.8, 95% confidence interval 1.6 to 28.1). CONCLUSIONS: Persistence of sialidase-producing vaginal microorganisms in numbers sufficient to increase vaginal fluid sialidase activity may be a risk factor for possibly preventable subclinical intrauterine infection and preterm birth. This study confirms and further informs our understanding of the association of bacterial vaginosis and preterm birth; studies to evaluate whether systemic treatment for bacterial vaginosis can effectively reduce vaginal mucolytic enzymes and risks of prematurity and other morbid outcomes are continuing.

L6 ANSWER 39 OF 87 EMBASE COPYRIGHT 2005 ELSEVIER INC. ALL RIGHTS RESERVED.
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ACCESSION NUMBER: 94263061 EMBASE
DOCUMENT NUMBER: 1994263061
TITLE: Vaginitis including bacterial vaginosis.
AUTHOR: Eschenbach D.A.
CORPORATE SOURCE: Department of Obstetrics/Gynecology, University of
Washington, Seattle, WA 98195, United States
SOURCE: Current Opinion in Obstetrics and Gynecology, (1994) 6/4
(389-391).
ISSN: 1040-872X CODEN: COOGEA
COUNTRY: United States
DOCUMENT TYPE: Journal; General Review
FILE SEGMENT: 004 Microbiology
010 Obstetrics and Gynecology
017 Public Health, Social Medicine and Epidemiology
037 Drug Literature Index
038 Adverse Reactions Titles
LANGUAGE: English
SUMMARY LANGUAGE: English

AB Bacterial vaginosis is a common lower genital tract infection.
Women with bacterial vaginosis have 100-1000 times more virulent bacterial
per ml of vaginal flora than women without this infection. This
tremendous increase in the concentration of bacteria has been recently
associated with postpartum and posthysterectomy infection and
preterm delivery.

L6 ANSWER 40 OF 87 BIOTECHDS COPYRIGHT 2005 THE THOMSON CORP. on STN
DUPLICATE 15

ACCESSION NUMBER: 1993-11517 BIOTECHDS
TITLE: Recombinant swine-pox virus capable of replication;
contains foreign DNA encoding antigen and/or a selectable
marker; use full as a recombinant vaccine
PATENT ASSIGNEE: Syntro
PATENT INFO: WO 9314194 22 Jul 1993
APPLICATION INFO: WO 1993-US324 13 Jan 1993
PRIORITY INFO: US 1992-820154 13 Jan 1992
DOCUMENT TYPE: Patent
LANGUAGE: English
OTHER SOURCE: WPI: 1993-243210 [30]

AB Recombinant swine-pox virus (SPV) S-SPV-003 (ATCC VR 2335), S-SPV-008
(ATCC VR 2339) and S-SPV-009 (ATCC 2344) are claimed. The SPVs repl
icate in an animal, when introduced as SPV DNA containing foreign DNA (I)
in an insertion site not required for replication (SPV thymidine-kinase
gene or AccI site within a HindIII-BglII fragment of SPV) and er the
control of an SPV promoter. (I) encodes an antigen (Ag) e.g.

pseudorabies virus glycoprotein 50 (A), glycoprotein-II, -III or -H, transmissible-gastroenteritis virus glycoprotein 195 or matrix protein, Se
 pig rota virus glycoprotein 38, pig parvo virus capsid protein, Se
 rpulina hyodysenteriae protective Ag, cattle diarrhea virus glycoprotein
 55, Newcastle-disease virus hemagglutinin-neuraminidase
 (B), pig influenza virus hemagglutinin or neuraminidase,
 foot-and-mouth-disease virus Ag, pig cholera virus Ag, African-pig-fever
 virus Ag or Mycoplasma hyopneumoniae Ag, especially (A) or (B) or a
 selectable marker (Escherichia coli beta-galactosidase (EC-3.2.1.23)). A
 vector, a recombinant SPV vaccine, a method for immunization and Vero or
 EMSK cells infected with SPV are also new. (100pp)

L6 ANSWER 41 OF 87 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation. on
 STN

ACCESSION NUMBER: 1993:523306 BIOSIS
 DOCUMENT NUMBER: PREV199396136713
 TITLE: Protection of mice from Sendai virus **infections**
 by recombinant vaccinia viruses: Effects of the inoculation
 routes on their replication sites in mice and induction of
 local respiratory immunity.
 AUTHOR(S): Takao, Shin-Ichi
 CORPORATE SOURCE: Dep. Bacteriol., Hiroshima Univ. Sch. Med., Hiroshima,
 Japan
 SOURCE: Medical Journal of Hiroshima University, (1993) Vol. 41,
 No. 4, pp. 243-257.
 CODEN: HDIZAB. ISSN: 0018-2087.
 DOCUMENT TYPE: Article
 LANGUAGE: Japanese
 ENTRY DATE: Entered STN: 19 Nov 1993
 Last Updated on STN: 19 Nov 1993

AB The present study was undertaken to examine protection of mice from Sendai
 virus **infections** by the recombinant vaccinia viruses expressing
 the hemagglutinin-neuraminidase and fusion proteins of Sendai
 virus, Vac-HN and Vac-F, respectively. Since vaccinia virus causes a
 systemic **infection** in animals, this study was especially focused
 on investigating effects of the inoculation routes (intraperitoneal and
 intranasal) on their replication sites in mice and induction of local
 respiratory immunity against Sendai virus. Vac-HN inoculated
 intraperitoneally was found to reach the lung and nasal turbinate.
 Immunohistochemistry of those organs showed, however, that its replication
 site was exclusively in the tooth germ, and that no vaccinia virus
 antigens were detected in the mucosal layer of respiratory tract where
 significant amounts of the antigens were detected of the intranasally
 inoculated mice. When Sendai virus was intranasally challenged to the
 mice immunized with either Vac-HN or Vac-F, replication of the challenged
 virus was almost completely suppressed both in the lung and in the nasal
 turbinate of the intranasally immunized mice, while the suppression was
 only partial in the lung of the intraperitoneally immunized mice, and
 nearly no suppression was observed in the nasal turbinate. The primary
 and secondary responses of Sendai virus-specific IgG and IgA antibodies and
 IgG- and IgA-producing cells were examined in the respiratory secretions
 and in the mucosal layer of nasal turbinate, respectively, in the mice
 immunized with either Vac-HN or Vac-F. The results showed that the
 secondary responses of the antibodies and cells evidently occurred only in
 the intranasally immunized mice on the challenge Sendai virus
infection, suggesting strongly that replication of the recombinant
 virus in the mucosal layer of respiratory tract is required for induction
 of the local respiratory immunity. The present study, therefore,
 indicates that the parenteral inoculation of the recombinant vaccinia
 virus is not enough to induce effective local respiratory immunity in mice
 because of its insufficient growth in the respiratory mucosal layer even
 though its pantropic nature.

L6 ANSWER 42 OF 87 SCISEARCH COPYRIGHT (c) 2005 The Thomson Corporation.

on STN
ACCESSION NUMBER: 92:616443 SCISEARCH
THE GENUINE ARTICLE: JT978
TITLE: IDENTIFICATION OF N-ACETYLLACTOSAMINE GLYCANS AS THE RECEPTORS OF
POLY-N-ACETYLLACTOSAMINE GLYCANS AS THE RECEPTORS OF
SIALIC ACID-BINDING STREPTOCOCCUS-SUIS STRAINS
AUTHOR: LIUKKONEN J; HAATAJA S; TIKKANEN K; KELM S; FINNE J
(Reprint)
CORPORATE SOURCE: UNIV TURKU, DEPT MED BIOCHEM, KIINAMYLLENKATU 10, SF-20520
TURKU 52, FINLAND; UNIV KUOPIO, DEPT BIOCHEM & BIOTECHNOL,
SF-70211 KUOPIO, FINLAND; UNIV CALIF LOS ANGELES, SCH MED,
DEPT BIOL CHEM, LOS ANGELES, CA, 90024; UNIV KIEL, INST
BIOCHEM, W-2300 KIEL 1, GERMANY
COUNTRY OF AUTHOR: FINLAND; USA; GERMANY
SOURCE: JOURNAL OF BIOLOGICAL CHEMISTRY, (15 OCT 1992) Vol. 267,
No. 29, pp. 21105-21111.
ISSN: 0021-9258.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE
LANGUAGE: ENGLISH
REFERENCE COUNT: 50

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Streptococcus suis is a common cause of sepsis, meningitis, and other serious infections in young piglets and also causes meningitis in humans. The cell-binding specificity of sialic acid-recognizing strains of Streptococcus suis was investigated. Treatment of human erythrocytes with sialidase or mild periodate abolished hemagglutination. Hemagglutination inhibition experiments with sialyl oligosaccharides indicated that the adhesin preferred the sequence NeuNAc α 2-3Gal β 1-4Glc(NAc). Resialylation of desialylated erythrocytes with Gal β 1-3(4)GlcNAc α 2-3-sialyltransferase induced a strong hemagglutination, whereas no or only weak hemagglutination was obtained with cells resialylated with two other sialyltransferases. Binding of radiolabeled bacteria to blots of erythrocyte membrane proteins revealed binding to the poly-N-acetyllactosamine-containing components Band 3, Band 4.5, and polyglycosyl ceramides and to glycophorin A. The involvement of glycophorin A as a major ligand was excluded by the strong hemagglutination of trypsin-treated erythrocytes and En(a-) erythrocytes defective in glycophorin A. Sensitivity of the hemagglutination toward endo-beta-galactosidase treatment of erythrocytes and inhibition by purified poly-N-acetyllactosaminyl glycopeptides indicated that the adhesin bound to glycans containing the following structure:
NeuNAc α 2-3Gal β 1-4GlcNAc β 1-3Gal β 1-

L6 ANSWER 43 OF 87 MEDLINE on STN DUPLICATE 16
ACCESSION NUMBER: 92104699 MEDLINE
DOCUMENT NUMBER: PubMed ID: 1370278
TITLE: Characterization of I/F1 glycoprotein as a receptor for
Mycoplasma pneumoniae.
AUTHOR: Hengge U R; Kirschfink M; Konig A L; Nicklas W; Roelcke D
CORPORATE SOURCE: Institute of Immunology, University of Heidelberg, Germany.
SOURCE: Infection and immunity, (1992 Jan) 60 (1) 79-83.
Journal code: 0246127. ISSN: 0019-9567.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199202
ENTRY DATE: Entered STN: 19920302
Last Updated on STN: 19970203
Entered Medline: 19920212

AB Serologic evidence of anti-I and anti-F1 cold agglutinins occurring in **mycoplasma infections** led to the isolation of I/F1 glycoprotein from human erythrocyte membranes. **Mycoplasma**

pneumoniae bound to purified I/Fl glycoprotein in a dose-dependent fashion depending on sialylated carbohydrate determinants. This was shown by the decreased binding of **mycoplasmas** to either **sialidase**-treated I/Fl glycoprotein (dot blot analysis) or **sialidase**-treated erythrocytes (hemagglutination test). Structural properties of the receptor for optimal binding could be explored by hemagglutination inhibition assays. Glycophorins were excluded as receptors. These results indicate that Fl (and I) antigens are receptors for *M. pneumoniae*.

L6 ANSWER 44 OF 87 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation. on STN

ACCESSION NUMBER: 1991:115202 BIOSIS
DOCUMENT NUMBER: PREV199191062592; BA91:62592
TITLE: ADHERENCE OF UREAPLASMA-UREALYTICUM TO HUMAN ERYTHROCYTES.
AUTHOR(S): SAADA A-B [Reprint author]; TERESPOLSKI Y; ADONI A; KAHANE I
CORPORATE SOURCE: DEP MEMBRANE, ULTRASTRUCTURE RES, HEBREW UNIV-HADASSAH MED SCH, JERUSALEM 91010
SOURCE: Infection and Immunity, (1991) Vol. 59, No. 1, pp. 467-469.
CODEN: INFIBR. ISSN: 0019-9567.
DOCUMENT TYPE: Article
FILE SEGMENT: BA
LANGUAGE: ENGLISH
ENTRY DATE: Entered STN: 27 Feb 1991
Last Updated on STN: 27 Feb 1991

AB *Ureaplasma urealyticum* (four serotypes and two clinical isolates) were metabolically labeled with radioactive methionine to a high specific activity. Labeling allowed the study of the mechanism of adherence to human erythrocytes. The adherence mechanism was complex and partially mediated by proteinaceous surface components. The binding sites on the erythrocytes were partially sensitive to **neuraminidase** treatment, and adherence was inhibited by glycophorin and dextran sulfate, indicating recognition of sialyl residues and sulfated compounds.

L6 ANSWER 45 OF 87 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation. on STN

ACCESSION NUMBER: 1991:5754 BIOSIS
DOCUMENT NUMBER: PREV199191005754; BA91:5754
TITLE: GLYCOSIDASE ACTIVITIES OF **MYCOPLASMAS**.
AUTHOR(S): KAHANE I [Reprint author]; REISCH-SAADA A; ALMAGOR M; ABELIUCK P; YATZIV S
CORPORATE SOURCE: DEP MEMBRANE ULTRASTRUCTURE RES, HEBREW UNIV-HADASSAH MED SCH, PO BOX 1172, JERUSALEM 91010, ISRAEL
SOURCE: Zentralblatt fuer Bakteriologie, (1990) Vol. 273, No. 3, pp. 300-305.
CODEN: ZEBAE8. ISSN: 0934-8840.
DOCUMENT TYPE: Article
FILE SEGMENT: BA
LANGUAGE: ENGLISH
ENTRY DATE: Entered STN: 8 Dec 1990
Last Updated on STN: 9 Dec 1990

AB The activities of α - and β -glucosidase, β -galactosidase and β -N acetylglucosaminidase were assessed at acidic pH by fluorimetry using the appropriate 4-methylumbelliferyl substrate in four **Mycoplasma** species (*M. pneumoniae*, *M. gallisepticum*, *M. hominis* and *M. capricolum*) and in *Acholeplasma laidlawii*. The glycosidase activities were in a low range (0.1-4.2 nmole per h per mg protein) with the exception of higher activities of β -N-acetylglucosaminidase in *A. laidlawii*. The enzyme levels of a virulent and a nonvirulent strain of *M. pneumoniae* were comparable. Despite the very sensitive assay, **neuraminidase** activity was not detected in *M. pneumoniae* and *M. gallisepticum*. No induction of α -glucosidase could be demonstrated for *M. pneumoniae* or *A. laidlawii*. At least part of the glucosidase activities was localized in the membrane fraction of all

mycoplasmas studied. This may support the hypothesis that pathogenic **mycoplasmas**, being membrane parasites, may modify, by their glycosidases, some host cell glycoconjugates. However, our study did not distinguish the pathogenic **mycoplasmas** to possess a characteristic glycosidase profile.

L6 ANSWER 46 OF 87 HCAPLUS COPYRIGHT 2005 ACS on STN

ACCESSION NUMBER: 1991:1768 HCAPLUS
 DOCUMENT NUMBER: 114:1768
 TITLE: A recombinant Marek's **disease** virus and its use as a live multifunctional vaccine
 INVENTOR(S): Ishikawa, Toyokazu; Manabe, Sadao; Mori, Chisato; Takamizawa, Akihisa; Yoshida, Iwao; Osame, Juichiro; Takaku, Keisuke; Fukai, Konosuke
 PATENT ASSIGNEE(S): Research Foundation for Microbial Diseases, Osaka University, Japan
 SOURCE: Eur. Pat. Appl., 59 pp.
 CODEN: EPXXDW
 DOCUMENT TYPE: Patent
 LANGUAGE: English
 FAMILY ACC. NUM. COUNT: 1
 PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
EP 334530	A1	19890927	EP 1989-302485	19890314
EP 334530	B1	19950118		
R: AT, BE, CH, DE, ES, FR, GB, GR, IT, LI, LU, NL, SE				
JP 02291277	A2	19901203	JP 1988-230851	19880914
JP 2779447	B2	19980723		
ES 2066842	T3	19950316	ES 1989-302485	19890314
CA 1340243	A1	19981215	CA 1989-593826	19890315
AU 8931428	A1	19890921	AU 1989-31428	19890317
AU 619812	B2	19920206		
HU 52555	A2	19900728	HU 1989-1319	19890320
US 5650153	A	19970722	US 1994-293337	19940824
PRIORITY APPLN. INFO.:			JP 1988-66973	A 19880320
			JP 1988-230851	A 19880914
			US 1989-324064	B1 19890316
			US 1992-948469	B1 19920922

AB Attenuated Marek's **disease** virus (MDV) and the related herpesvirus of turkey (HVT) are used to express genes for heterologous antigens in avian systems. This allows their use as a general-purpose live vaccine for domestic fowl. Genes for antigens of HVT and Newcastle **disease** virus (the hemagglutinin-neuraminidase gene) were cloned into attenuated HVT and MDV and recombinant virus used to vaccinate chickens. After antibody titers had been established all chickens challenged with the appropriate virus were resistant. All animals in the control groups died upon **infection** with a comparable inoculum.

L6 ANSWER 47 OF 87 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation.. on STN

ACCESSION NUMBER: 1989:382654 BIOSIS
 DOCUMENT NUMBER: PREV198988063244; BA88:63244
 TITLE: HEMAGGLUTINATION AND HEMAGGLUTINATION INHIBITION OF TURKEY RED BLOOD CELLS WITH **MYCOPLASMA**-HYOPNEUMONIAE.
 AUTHOR(S): YOUNG T F [Reprint author]; ERICKSON B Z; ROSS R F; WANNEMUEHLER Y
 CORPORATE SOURCE: VET MED RES INST, IOWA STATE UNIV, AMES, IOWA 50011, USA
 SOURCE: American Journal of Veterinary Research, (1989) Vol. 50, No. 7, pp. 1052-1055.
 CODEN: AJVRAH. ISSN: 0002-9645.
 DOCUMENT TYPE: Article

FILE SEGMENT: BA
LANGUAGE: ENGLISH
ENTRY DATE: Entered STN: 17 Aug 1989
Last Updated on STN: 17 Aug 1989

AB The ability of **Mycoplasma** hyopneumoniae to agglutinate RBC was evaluated to develop an in vitro cytoadsorption assay. Using swine RBC in a microtitration hemagglutination test, no agglutination or partial agglutination was detected. Comparison of RBC from various other species indicated that improved hemagglutination was obtained with RBC from turkeys. This hemagglutination was detected only when **mycoplasma** cells used in the assay had been frozen and thawed, heated at 50 C for 30 minutes, or treated with trypsin. Treatment of RBC with trypsin or **neuraminidase** enhanced hemagglutination. Possible surface lectin activity in M hyopneumoniae was evaluated by use of carbohydrates in a blocking assay; hemagglutination was not inhibited by any of 13 carbohydrates evaluated. **Mycoplasma** hyopneumoniae convalescent porcine serum and monoclonal antibodies against 2 M hyopneumoniae immunogens of molecular weights of 64,000 and 41,000 inhibited hemagglutination.

L6 ANSWER 48 OF 87 MEDLINE on STN
ACCESSION NUMBER: 88032236 MEDLINE
DOCUMENT NUMBER: PubMed ID: 3312097
TITLE: Attachment of **Mycoplasma** hominis to human cell cultures.
COMMENT: Erratum in: Isr J Med Sci 1987 Aug;23(8):preceding 869
AUTHOR: Izumikawa K; Chandler D K; Grabowski M W; Barile M F
CORPORATE SOURCE: Sasebo General Hospital, Japan.
SOURCE: Israel journal of medical sciences, (1987 Jun) 23 (6) 603-7.
Journal code: 0013105. ISSN: 0021-2180.
PUB. COUNTRY: Israel
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198712
ENTRY DATE: Entered STN: 19900305
Last Updated on STN: 19970203
Entered Medline: 19871215

AB Clinical isolates, cell-culture contaminants, and the type strain PG21 of **Mycoplasma** hominis were examined for attachment to erythrocytes and human cell cultures. Strain 13428 (from blood, postpartum fever) and strain 1184 (cell culture) attached to human and guinea pig erythrocytes, but there were no differences in attachment activities between these strains. However, five M. hominis strains isolated from different tissue sites showed quantitative differences in attachment to human WiDr (intestinal carcinoma cell cultures), MRC-5 (human embryonic lung fibroblasts) and HeLa (carcinoma of cervix) cell cultures. The relative attachment activities were, in descending order: strain 1184 (cell culture), strain 11932 (cervix), strain 13428 (blood, postpartum fever), 13408 (nongonococcal urethritis), and type strain PG21 (multiple passage, originally from human rectum). Trypsin and pronase treatment of M. hominis strain 1184 markedly reduced attachment, suggesting that surface proteins play a role in M. hominis attachment to mammalian cells. In subsequent studies, strain 1620 (septic arthritis) showed the highest attachment activity, whereas strain 1652 (surgical skin flap) and L01888 (cell culture) showed attachment activity similar to cell culture strain 1184. The differing attachment activities of these M. hominis strains isolated from different infected sites of patients with a variety of diseases may be relevant to the virulence of these strains.

L6 ANSWER 49 OF 87 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation. on STN
ACCESSION NUMBER: 1988:216355 BIOSIS

DOCUMENT NUMBER: PREV198834109365; BR34:109365
TITLE: INTERACTIONS OF **MYCOPLASMA**-BOVOCULI STRAINS WITH
ERYTHROCYTES.
AUTHOR(S): SALIH B A [Reprint author]; ROSENBUSCH R F
CORPORATE SOURCE: IOWA STATE UNIV, AMES, IOWA, USA
SOURCE: Israel Journal of Medical Sciences, (1987) Vol. 23, No. 5,
pp. 516.
Meeting Info.: SIXTH INTERNATIONAL CONGRESS OF THE
INTERNATIONAL ORGANIZATION FOR MYCOPLASMOLOGY, BIRMINGHAM,
ALABAMA, USA, AUGUST 26-31, 1986. ISR J MED SCI.
CODEN: IJMDAI. ISSN: 0021-2180.
DOCUMENT TYPE: Conference; (Meeting)
FILE SEGMENT: BR
LANGUAGE: ENGLISH
ENTRY DATE: Entered STN: 25 Apr 1988
Last Updated on STN: 25 Apr 1988

L6 ANSWER 50 OF 87 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation. on
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ACCESSION NUMBER: 1988:113383 BIOSIS
DOCUMENT NUMBER: PREV198885058853; BA85:58853
TITLE: INTERACTION OF **MYCOPLASMA**-MOBILE 163K WITH
ERYTHROCYTES.
AUTHOR(S): FISCHER M [Reprint author]; KIRCHHOFF H
CORPORATE SOURCE: INST FUER MIKROBIOL UND TIERSEUCHEN DER TIERAERZTLICHEN
HOCHSCHULE, BISCHOFSHOLER DAMM 15, D-3000 HANNOVER
SOURCE: Zentralblatt fuer Bakteriologie Mikrobiologie und Hygiene
Series A, (1987) Vol. 266, No. 3-4, pp. 497-505.
CODEN: ZBMPEJ. ISSN: 0176-6724.
DOCUMENT TYPE: Article
FILE SEGMENT: BA
LANGUAGE: ENGLISH
ENTRY DATE: Entered STN: 23 Feb 1988
Last Updated on STN: 23 Feb 1988

AB **Mycoplasma** (M.) mobile 163K isolated from fish was investigated
for its hemadsorbing, hemagglutinating, and hemolysing capacities and for
its ability to adhere to erythrocytes. Hemadsorption to the colonies of
M. mobile occurred with ovine, bovine, equine, trout, and carp
erythrocytes and was inhibited by treatment of the **mycoplasmas**
with substances acting on proteins (pronase, trypsin, glutaraldehyde),
heat, UV-irradiation and homologous antiserum. Hemadsorption could be
prevented also by treatment of the erythrocytes with **neuraminidase**
. In liquid medium ovine erythrocytes were agglutinated and afterwards
lysed by M. mobile. The erythrocytes which were adsorbed to the colonies
of M. mobile were finally lysed also. Darkfield preparations showed the
ability of M. mobile to adhere to erythrocytes and also its
hemagglutinating properties.

L6 ANSWER 51 OF 87 MEDLINE on STN DUPLICATE 17

ACCESSION NUMBER: 86149426 MEDLINE
DOCUMENT NUMBER: PubMed ID: 3081908
TITLE: **Mycoplasma pneumoniae** attachment to
glutaraldehyde-treated human WiDr cell cultures.
AUTHOR: Izumikawa K; Chandler D K; Barile M F
SOURCE: Proceedings of the Society for Experimental Biology and
Medicine. Society for Experimental Biology and Medicine
(New York, N. Y.), (1986 Apr) 181 (4) 507-11.
Journal code: 7505892. ISSN: 0037-9727.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198604
ENTRY DATE: Entered STN: 19900321

Last Updated on STN: 19900321

Entered Medline: 19860418

AB Attachment of *Mycoplasma pneumoniae* to host cells initiates **disease**, and the attachment components may represent important protective immunogens for preventing **disease**. We have studied the mechanisms of attachment using in vitro cell culture systems and selected pathogenic and nonpathogenic strains of *M. pneumoniae*. Attachment of the pathogenic strains M129 and PI-1428 was several fold greater than attachment of the nonpathogenic strain, and attachment of strains M129 and PI-1428 was reduced by 21 to 63% when human WiDr cell monolayers were exposed to **neuraminidase**, supporting the concept that *M. pneumoniae* attaches to mammalian cells by a **neuraminidase**-sensitive glycoconjugate. While attachment of the two pathogenic strains was markedly reduced by treating the WiDr cells with glutaraldehyde, glutaraldehyde treatment produced minimal effects on the attachment of the nonpathogenic strain B176. Glutaraldehyde treatment also altered the temperature dependence of attachment by the pathogenic strains. Because glutaraldehyde-treated WiDr cell monolayers showed little difference in attachment between pathogenic and nonpathogenic strains, glutaraldehyde-treated cells are not appropriate cell substrates for studying *M. pneumoniae* attachment mechanisms or identifying immunogens for vaccine development.

L6 ANSWER 52 OF 87 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation. on STN

ACCESSION NUMBER: 1986:281156 BIOSIS

DOCUMENT NUMBER: PREV198682025019; BA82:25019

TITLE: DETECTION AND DIFFERENTIATION OF **MYCOPLASMA**
-GALLISEPTICUM AND **MYCOPLASMA**-SYNOVIAE ANTIBODIES
IN CHICKEN SERUM USING ELISA.

AUTHOR(S): HIGGINS P A [Reprint author]; WHITHEAR K G

CORPORATE SOURCE: DEPARTMENT VETERINARY PARACLINICAL SCIENCES, UNIVERSITY
MELBOURNE PARKVILLE, VICTORIA 3052, AUSTRALIA

SOURCE: Avian Diseases, (1986) Vol. 30, No. 1, pp. 160-168.
CODEN: AVDIAI. ISSN: 0005-2086.

DOCUMENT TYPE: Article

FILE SEGMENT: BA

LANGUAGE: ENGLISH

ENTRY DATE: Entered STN: 4 Jul 1986

Last Updated on STN: 4 Jul 1986

AB Affinity-purified sheep IgG anti-chicken IgG horseradish peroxidase conjugate was utilized in an enzyme-linked immunosorbent assay (ELISA) to detect *Mycoplasma gallisepticum*- and *M. synoviae*-specific antibodies in chicken sera. Antigen, conjugate and substrate concentrations, and incubation times were adjusted to provide maximum differentiation between positive and negative sera. Use of phosphate-buffered saline containing 0.05% Tween 20 for washing and diluting steps and use of normal sheep serum to make the initial 1:10 serum dilution resulted in optimal differentiation between homologous and heterologous antisera. However, sera known to contain antibodies to *M. gallisepticum* or *M. synoviae* gave higher absorbance values with the heterologous antigen than did specific-pathogen-free sera. To reduce the frequency of nonspecific reactions to less than 2% it was necessary to adjust the threshold absorbance for each antigen according to the known infectious status of the flock. Reproducibility of the assay was maintained by using positive and negative control sera on each plate. Results from 14.2% of the plates tested were rejected, because the endpoint of the positive control serum was more than one dilution from the most common value. Of four strains of *M. gallisepticum* used as antigens, none was clearly superior to the others in producing maximum titers with a range of *M. gallisepticum* antisera. However, nonspecific absorbance tended to be less with the S6 strain. The stability of *M. gallisepticum*-coated plates was maintained for up to 6 months at -8 C or below, whereas *M. synoviae*-coated plates were stored satisfactorily for 6

months at 4 C or below. No correlation could be found between the nonspecific absorbance reading of an individual serum and its absorbance to bovine IgG or its specific ELISA titer. Nonspecific reactions were not reduced by heat inactivation, mercaptoethanol or **neuraminidase** treatment, or delipidization of serum.

L6 ANSWER 53 OF 87 MEDLINE on STN
ACCESSION NUMBER: 86108757 MEDLINE
DOCUMENT NUMBER: PubMed ID: 3943586
TITLE: Anaplasma marginale, Eperythrozoon wenyonii: lectin reactions with bovine erythrocytes.
AUTHOR: Goff W L; Johnson L W; Kuttler K L
SOURCE: Experimental parasitology, (1986 Feb) 61 (1) 103-13.
Journal code: 0370713. ISSN: 0014-4894.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198603
ENTRY DATE: Entered STN: 19900321
Last Updated on STN: 19980206
Entered Medline: 19860312

AB Normal bovine erythrocytes were agglutinated with four of five lectins specific for different oligosaccharides. The order of reactivity was wheat germ greater than ricin greater than soybean greater than peanut. Concanavalin A did not agglutinate normal bovine erythrocytes. After **neuraminidase** treatment of normal bovine erythrocytes, each lectin agglutinated the cells with decreased concentrations of lectin, verifying that partial removal of sialic acid exposes more of each lectin's binding sites or alters the binding site such that fewer molecules of lectin are required to initiate agglutination. A change in agglutination of erythrocytes using soybean agglutinin and peanut agglutinin occurred when cells were obtained from cattle infected with Eperythrozoon wenyonii. The results suggested that an alteration in erythrocyte membranes occurred as a result of this **infection** as manifested by the increased recognition of both the soybean agglutinin and peanut agglutinin receptor carbohydrates. A similar effect was indicated with erythrocytes obtained during an acute Anaplasma marginale **infection**; however, an ensuing reticulocytosis masked the effect, requiring the use of fluoresceinated lectins to verify that increased binding of each lectin occurred with infected cells when compared to normal cells.

L6 ANSWER 54 OF 87 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation. on STN
ACCESSION NUMBER: 1985:426276 BIOSIS
DOCUMENT NUMBER: PREV198580096268; BA80:96268
TITLE: ATTACHMENT OF MYCOPLASMA-PULMONIS TO RAT AND MOUSE SYNOVIAL CELLS CULTURED IN-VITRO.
AUTHOR(S): ARAAKE M [Reprint author]; YAYOSHI M; YOSHIOKA M
CORPORATE SOURCE: DEP OF MICROBIOLOGY, TOKYO WOMEN'S MED COLL, KAWADA-CHO, SHINJUKU-KU, TOKYO 162
SOURCE: Microbiology and Immunology, (1985) Vol. 29, No. 7, pp. 601-608.
CODEN: MIIMDV. ISSN: 0385-5600.
DOCUMENT TYPE: Article
FILE SEGMENT: BA
LANGUAGE: ENGLISH

AB The attachment of M. pulmonis m53 organisms to mouse and rat synovial cells was examined by using the organisms and the synovial cells treated in various ways. M. pulmonis treated with trypsin attached to the synovial cells, but the organisms treated with pronase, formaldehyde, glutaraldehyde, or heat did not. These findings suggest that the sites for binding M. pulmonis to the mouse and rat synovial cells are of polypeptide nature. Treatment of M. pulmonis with sialic acid and

treatment of the synovial cell sheets with **neuraminidase** did not affect the attachment. The synovial cell surface for receptors M. pulmonis organisms would be different from those on respiratory cells or erythrocytes for M. pneumoniae or M. gallisepticum. Even nonviable organisms and M. pulmonis membranes attached to the mouse or rat synovial cells. The nature of the receptor of mouse synovial cells would be different from that of rat cells, since rat cells were affected by treatment with formaldehyde or glutaraldehyde, but mouse cells were not.

L6 ANSWER 55 OF 87 EMBASE COPYRIGHT 2005 ELSEVIER INC. ALL RIGHTS RESERVED.
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ACCESSION NUMBER: 85023914 EMBASE
DOCUMENT NUMBER: 1985023914
TITLE: Autoimmune hemolytic anemia by coexisting anti-I and anti-F1 cold agglutinins.
AUTHOR: Konig A.L.; Kather H.; Roelcke D.
CORPORATE SOURCE: Institute for Immunology and Serology, University of Heidelberg, D-6900 Heidelberg, Germany
SOURCE: Blut, (1984) 49/5 (363-368).
CODEN: BLUTA
COUNTRY: Germany
DOCUMENT TYPE: Journal
FILE SEGMENT: 025 Hematology
026 Immunology, Serology and Transplantation
LANGUAGE: English

AB In association with atypical pneumonia, a patient developed acute severe autoimmune hemolytic anemia. Hemoglobin temporarily was only 7.0 g/100 ml, so that the patient needed red blood cell (RBC) transfusion. Hemolysis was found to be caused by high titer cold agglutinins (CA), which occurred transiently during the acute period of the **disease**. CA of two different specificities, anti-I and anti-F1, were demonstrated in the patient's serum. Antibodies of the two specificities were clearly separated by absorption/elution experiments using **neuraminidase** (RDE)-treated RBC. They were distinguished by serologic means: Both anti-I and anti-F1 react more strongly with adult RBC than with newborn and adult RBC; in contrast to anti-I, anti-F1 does not agglutinate RDE-treated cells. Inhibition experiments showed that I-active substances prepared from papainized RBC exhibited both I and F1 antigenic activity. By RDE-treatment of I-active substances, F1-activity was markedly reduced, while I-activity was increased.

L6 ANSWER 56 OF 87 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation. on STN

ACCESSION NUMBER: 1983:253856 BIOSIS
DOCUMENT NUMBER: PREV198376011348; BA76:11348
TITLE: HEM ADSORPTION AND VIRULENCE ARE SEPARABLE PROPERTIES OF **MYCOPLASMA-PNEUMONIAE**.
AUTHOR(S): LEITH D K [Reprint author]; HANSEN E J; WILSON R M; KRAUSE D C; BASEMAN J B
CORPORATE SOURCE: DEP MICROBIOL, UNIV TEX HEALTH SCI CENT, SAN ANTONIO, TEX 78248, USA
SOURCE: Infection and Immunity, (1983) Vol. 39, No. 2, pp. 844-850.
CODEN: INFIBR. ISSN: 0019-9567.
DOCUMENT TYPE: Article
FILE SEGMENT: BA
LANGUAGE: ENGLISH

AB A selective enrichment technique was used to isolate a hemadsorption-positive revertant of a hemadsorption-negative mutant strain of M. pneumoniae. This hemadsorption-positive revertant simultaneously regained both the ability to attach to **neuraminidase**-sensitive receptors on the tracheal ring respiratory epithelium in vitro and the ability to synthesize 3 virulent-strain-specific proteins which were not synthesized by the hemadsorption-negative mutant. Despite the persistence of the revertant in hamster lung tissue for 9-12 wk postinfection, no

cytopathology was observed. Intranasal inoculation of the revertant provided limited protection against a challenge dose of virulent *M. pneumoniae*.

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ACCESSION NUMBER: 1983:253854 BIOSIS
DOCUMENT NUMBER: PREV198376011346; BA76:11346
TITLE: REACQUISITION OF SPECIFIC PROTEINS CONFERS VIRULENCE IN **MYCOPLASMA-PNEUMONIAE**.
AUTHOR(S): KRAUSE D C [Reprint author]; LEITH D K; BASEMAN J B
CORPORATE SOURCE: DEP MICROBIOL, UNIV TEX HEALTH SCI CENT, SAN ANTONIO, TEX 78284, USA
SOURCE: Infection and Immunity, (1983) Vol. 39, No. 2, pp. 830-836. CODEN: INFIBR. ISSN: 0019-9567.
DOCUMENT TYPE: Article
FILE SEGMENT: BA
LANGUAGE: ENGLISH

AB Hemadsorbing revertants were isolated from spontaneous hemadsorption-negative, avirulent mutants of *M. pneumoniae*. The revertants simultaneously reacquired specific proteins absent in their homologous mutants, along with **neuraminidase**-sensitive adherence to the hamster respiratory epithelium and virulence. Peptide mapping and immunological analysis indicated no precursor-product relationships among certain of these proteins.

L6 ANSWER 58 OF 87 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation. on STN

ACCESSION NUMBER: 1982:238392 BIOSIS
DOCUMENT NUMBER: PREV198274010872; BA74:10872
TITLE: IDENTIFICATION OF **MYCOPLASMA-PNEUMONIAE** PROTEINS ASSOCIATED WITH HEM ADSORPTION AND VIRULENCE.
AUTHOR(S): KRAUSE D C [Reprint author]; LEITH D K; WILSON R M; BASEMAN J B
CORPORATE SOURCE: DEP MICROBIOL, UNIV TEXAS HEALTH SCI CENT, SAN ANTONIO, TEX 78284, USA
SOURCE: Infection and Immunity, (1982) Vol. 35, No. 3, pp. 809-817. CODEN: INFIBR. ISSN: 0019-9567.
DOCUMENT TYPE: Article
FILE SEGMENT: BA
LANGUAGE: ENGLISH

AB Twenty-two mutants of *M. pneumoniae* spontaneously deficient in hemadsorption were isolated. Examination of mutant protein profiles by 1- and 2-dimensional polyacrylamide gel electrophoresis permitted the grouping of these mutants into 4 classes. The largest class of mutants was deficient in 4 high-MW proteins (215,000, 210,000, 190,000 and 140,000). A 2nd class of mutants lacked 3 proteins previously designated A, B and C (72,000, 85,000 and 37,000, respectively). A single mutant, in addition to lacking proteins A, B and C, was missing a 4th protein of 165,000 MW. The remaining mutants exhibited protein profiles apparently identical to that of the wild-type strain. All mutant strains attached to the respiratory epithelium of hamster tracheal rings in vitro at reduced levels; however, mutants lacking proteins A, B and C recognized only **neuraminidase**-insensitive receptors. None of the mutants tested produced detectable pneumonia in intranasally inoculated hamsters, although 1 mutant class demonstrated low-level survival in vivo.

L6 ANSWER 59 OF 87 MEDLINE on STN DUPLICATE 18

ACCESSION NUMBER: 83016243 MEDLINE
DOCUMENT NUMBER: PubMed ID: 6812201
TITLE: A review of the morphological and biochemical features of the attachment process in **infections** with **Mycoplasma pneumoniae**.
AUTHOR: Gabridge M G

CONTRACT NUMBER: AI 12559 (NIAID)

HL 23806 (NHLBI)

SOURCE: Reviews of infectious diseases, (1982 May-Jun) 4 Suppl
S179-84.

Journal code: 7905878. ISSN: 0162-0886.

PUB. COUNTRY: United States

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English

FILE SEGMENT: Priority Journals

ENTRY MONTH: 198212

ENTRY DATE: Entered STN: 19900317

Last Updated on STN: 19970203

Entered Medline: 19821202

AB **Mycoplasma pneumoniae** must attach to respiratory tract cells to cause primary atypical pneumoniae. The attachment process involves a receptor site on the external membrane surface of the host cell and a specialized attachment tip on the mycoplasmal cells. Attachment to lung fibroblasts and ciliated tracheal explants is time dependent, with maxima reached in 45-90 min at 37 C. Attachment to ciliated cells is slower, apparently because of continuous ciliary motion. Normally, less than 10% of available **mycoplasmas** become cell associated in vitro, perhaps because the pathogen must be in a particular growth phase or because only a small fraction of the *M. pneumoniae* population has complete or effective attachment tips. **Mycoplasmas** that attach to host cells normally have the constricted attachment tip oriented toward the host cell surface. **Mycoplasmas** are oriented vertically in cultures of densely ciliated cells, but can lie horizontally alone--and in close apposition to--cell membranes of sparsely ciliated or nonciliated cells. The site to which *M. pneumoniae* attaches, a sialoglycoprotein, is readily inactivated by **neuraminidase**, partially sensitive to pronase, and resistant to trypsin. Purified glycoprotein extracts bind to *M. pneumoniae*.

L6 ANSWER 60 OF 87 MEDLINE on STN DUPLICATE 19

ACCESSION NUMBER: 82074432 MEDLINE

DOCUMENT NUMBER: PubMed ID: 6796499

TITLE: **Mycoplasma pneumoniae infection of**
intact guinea pig tracheas cultured in a unique
matrix-embed/perfusion system.

AUTHOR: Gabridge M G; Hoglund L E

CONTRACT NUMBER: AI 12559 (NIAID)

AI 17795 (NIAID)

HL 23806 (NHLBI)

SOURCE: In vitro, (1981 Oct) 17 (10) 847-58.

Journal code: 0063733. ISSN: 0073-5655.

PUB. COUNTRY: United States

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English

FILE SEGMENT: Priority Journals

ENTRY MONTH: 198202

ENTRY DATE: Entered STN: 19900316

Last Updated on STN: 19970203

Entered Medline: 19820212

AB A new method for the in vitro culture of entire, intact tracheas from adult guinea pigs is described. Matrix-embed/perfusion (MEP) culture is based on an immobilization of the tissue in nutrient agar. The tubular piece of agar-embedded organ was contained in a special perfusion block with two wells for liquid medium at either end. When incubated on a rocker platform, liquid medium flows through the trachea and supplies oxygen and nutrients. In this configuration, tracheas maintain near-normal metabolism (ATP content and dehydrogenase activity), structure (as determined by light and electron microscopy), and function (ciliary motion). Tissues could be maintained in vitro in a normal for at least 4 wk, with reduced ciliary motion and cell metabolism detectable for at

least 6 wk. Agar-embedded tissues from the MEP cultures were nearly identical to those cultivated with standard tracheal ring explant techniques. Tracheas in the MEP cultures were infected with **Mycoplasma pneumoniae**. Attachment was **neuraminidase**-sensitive. **Mycoplasma** attachment was lowest on the epithelium along the dorsal ridge, but was uniform along the length of the trachea. Ciliostasis and cytonecrosis induced by **M. pneumoniae** was dose dependent. The matrix-embed/perfuse technique appears to have considerable potential for several types of in vitro studies on trachea or other tubular organs.

L6 ANSWER 61 OF 87 MEDLINE on STN DUPLICATE 20
ACCESSION NUMBER: 81167005 MEDLINE
DOCUMENT NUMBER: PubMed ID: 6163719
TITLE: Characterization of hemadsorption-negative mutants of **Mycoplasma pneumoniae**.
AUTHOR: Hansen E J; Wilson R M; Clyde W A Jr; Baseman J B
CONTRACT NUMBER: 1-KO4-AI-00178 (NIAID)
HL-19171 (NHLBI)
SOURCE: Infection and immunity, (1981 Apr) 32 (1) 127-36.
Journal code: 0246127. ISSN: 0019-9567.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198106
ENTRY DATE: Entered STN: 19900316
Last Updated on STN: 19970203
Entered Medline: 19810623

AB Previously isolated mutants of **Mycoplasma pneumoniae** incapable of hemadsorption were characterized with respect to specific protein content, tracheal ring attachment capability, and virulence for both in vitro and in vivo model systems. Two-dimensional gel electrophoresis revealed both quantitative and qualitative differences between the protein complements of two different mutant strains and that of the virulent parent strain. Studies of **mycoplasma** attachment to hamster tracheal rings in vitro demonstrated that only one of these mutant strains still possessed the ability to attach to the respiratory epithelium via **neuraminidase**-sensitive receptors. Measurement of [³H]orotic acid uptake in **mycoplasma**-infected tracheal rings indicated that **infection** with the hemadsorption-negative mutants resulted in only slight reductions of ribonucleic acid synthesis, similar to levels observed for tracheal rings infected with an avirulent strain of **M. pneumoniae**. The virulence potential of the two mutant strains was further investigated by utilizing the hamster model system. Both mutant strains were rapidly cleared from the lungs of infected animals and produced little or no microscopic pneumonia.

L6 ANSWER 62 OF 87 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation. on STN
ACCESSION NUMBER: 1981:169197 BIOSIS
DOCUMENT NUMBER: PREV198171039189; BA71:39189
TITLE: INTERACTION OF **MYCOPLASMA**-GALLISEPTICUM WITH SIALYL GLYCO PROTEINS.
AUTHOR(S): GLASGOW L R [Reprint author]; HILL R L
CORPORATE SOURCE: DEP OF MED, VETERANS ADMINISTRATION HOSP, DURHAM, NC 27710, USA
SOURCE: Infection and Immunity, (1980) Vol. 30, No. 2, pp. 353-361.
CODEN: INFIBR. ISSN: 0019-9567.
DOCUMENT TYPE: Article
FILE SEGMENT: BA
LANGUAGE: ENGLISH

AB The binding of several glycoproteins to freshly grown and harvested cells of **M. gallisepticum** was examined. Only human glycophorin, the major sialoglycoprotein of the erythrocyte membrane, bound tightly as judged by

direct binding assays with 125I-labeled glycoproteins. **Neuraminidase**-treated glycophorin did not bind, suggesting that binding is mediated through sialic acid groups. Although other sialoglycoproteins did not appear to bind *M. gallisepticum* by direct binding assays, some inhibited the binding of glycophorin. The best inhibitors had a mucin-like structure, with high MW and high sialic acid contents. N-acetylneuraminic acid appeared to be the favored sialic acid structure for binding but there was no strict specificity for its anomeric linkage. **Neuraminidase** activity could not be detected on the surface of *M. gallisepticum*, suggesting that this enzyme is not involved in the mechanism of adherence of sialoglycoproteins. Binding of sialoglycoproteins was time dependent and markedly diminished with increasing ionic strength but was largely unaffected between pH 4-9.

L6 ANSWER 63 OF 87 EMBASE COPYRIGHT 2005 ELSEVIER INC. ALL RIGHTS RESERVED. on STN

ACCESSION NUMBER: 80142806 EMBASE
DOCUMENT NUMBER: 1980142806
TITLE: **Mycoplasmas** and ureaplasmas in infertility and abortion.
AUTHOR: Friberg J.
CORPORATE SOURCE: Dept. Obstet. Gynecol., Downstate Med. Cent., Brooklyn, N.Y. 11203, United States
SOURCE: Fertility and Sterility, (1980) 33/4 (351-359).
CODEN: FESTAS
COUNTRY: United States
DOCUMENT TYPE: Journal
FILE SEGMENT: 037 Drug Literature Index
010 Obstetrics and Gynecology
004 Microbiology
LANGUAGE: English

AB In several of the reports the highest conception rates were obtained in couples with unexplained infertility treated with appropriate antibiotics following a positive ureaplasma culture. The great variability in the pregnancy results may be due to a multifactorial etiology for couples considered to have 'unexplained infertility'. A prominent role for ureaplasmas in infertility should not be expected, although some of the data are quite suggestive. A number of investigators firmly believe that ureaplasmas are of importance in infertility, whereas others do not share this view. Genital ureaplasmas have been demonstrated in a large proportion of fertile couples and therefore it has been proposed that only specific strains of urea-plasmas might be causing infertility, perhaps by secretion of specific substances such as **neuraminidase**, ammonia, or other 'toxic factors' that may inhibit conception and/or disturb development of the embryo with the risk of subsequent abortion. Some ureaplasmas in the female genital tract may also adversely affect the function of the tubal epithelium with destruction of the cilia. Serotyping of ureaplasmas might give additional information, but it has also been suggested that the **infection** is only superficial and a systemic antibody response might not be indicative of a current **infection**. For years it has been thought that **mycoplasmas** and ureaplasmas were species-specific and therefore Koch's postulates about an infecting organism could not be carried out. However, it has been recently demonstrated that the chimpanzee can be infected with human ureaplasmas. It is also possible that other, less expensive, subhuman primates can be similarly inoculated. The development of a suitable animal model could provide a valuable new approach for the study of ureaplasmas in human infertility.

L6 ANSWER 64 OF 87 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation. on STN

ACCESSION NUMBER: 1980:128717 BIOSIS
DOCUMENT NUMBER: PREV198069003713; BA69:3713
TITLE: INTERACTION OF **MYCOPLASMA**-PNEUMONIAE WITH HUMAN

LUNG FIBROBLASTS ROLE OF RECEPTOR SITES.

AUTHOR(S): GABRIDGE M G [Reprint author]; TAYLOR-ROBINSON D
 CORPORATE SOURCE: SCH BASIC MED SCI, UNIV ILL, URBANA, ILL 61801, USA
 SOURCE: Infection and Immunity, (1979) Vol. 25, No. 1, pp. 455-459.
 CODEN: INFIBR. ISSN: 0019-9567.
 DOCUMENT TYPE: Article
 FILE SEGMENT: BA
 LANGUAGE: ENGLISH

AB The biochemical nature of the **neuraminidase**-sensitive M. pneumoniae receptor site on human lung fibroblast cells was studied. Purified, mixed sialoglycolipid (ganglioside) preparations from human and bovine tissues did not bind to M. pneumoniae organisms and block their subsequent attachment to fibroblasts. Fibroblasts incubated for 24 h in sialoglycolipid solutions to increase the ganglioside content of their membranes did not show increased pathogen attachment when later incubated with **mycoplasmas**. HeLa [human cervical carcinoma] cells grown in the presence of sodium butyrate to increase GM3 ganglioside levels did not have significantly increased uptake of M. pneumoniae organisms. Treatment of fibroblasts with enzymes indicated that the **mycoplasma** receptor site is trypsin- and papain-resistant but pronase-sensitive. Pronase digests of fibroblast membranes contained a product(s) which combined with M. pneumoniae cells and co-sedimented with them during centrifugation. Glycoproteins purified from fibroblast membranes by a lithium diiodosalicylate solubilization technique bound to M. pneumoniae organisms. The major component of the M. pneumoniae receptor site may be a sialoglycoprotein with little or no lipid.

L6 ANSWER 65 OF 87 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation. on STN

ACCESSION NUMBER: 1980:128716 BIOSIS
 DOCUMENT NUMBER: PREV198069003712; BA69:3712
 TITLE: INTERACTION OF **MYCOPLASMA**-PNEUMONIAE WITH HUMAN LUNG FIBROBLASTS CHARACTERIZATION OF THE IN-VITRO MODEL.
 AUTHOR(S): GABRIDGE M G [Reprint author]; TAYLOR-ROBINSON D; DAVIES H A; DOURMASHKIN R R
 CORPORATE SOURCE: SCH BASIC MED SCI, UNIV ILL, URBANA, ILL 61801, USA
 SOURCE: Infection and Immunity, (1979) Vol. 25, No. 1, pp. 446-454.
 CODEN: INFIBR. ISSN: 0019-9567.
 DOCUMENT TYPE: Article
 FILE SEGMENT: BA
 LANGUAGE: ENGLISH

AB The interaction of pathogenic M. pneumoniae and host cells was studied in cell cultures of MRC-5 human lung fibroblasts. A comparison of results obtained with fibroblasts in a monolayer format and with hamster tracheal explant cultures indicated that the former can bind significantly larger numbers of **mycoplasmas**. The attachment was 96% specific, i.e., mediated through a **neuraminidase**-sensitive receptor on the host cell. Uptake of **mycoplasmas** was directly related to the number of **Mycoplasma** cells present in the inoculum, and attachment was virtually complete within a 30-min period at 37° C. High doses of M. pneumoniae induced a marked cytopathic effect whereas doses of ≤ 10⁶ colony-forming units/ml produced grossly observable cell damage that was moderate and variable. Transmission electron microscopy studies indicated that attachment of M. pneumoniae to the surface of lung fibroblasts occurred with the specialized terminal structure or binding site oriented closest to the epithelial cell surface. The filamentous **Mycoplasma** cells were spatially arranged in several configurations and were not limited to a vertical orientation. The advantages and disadvantages of human lung fibroblast monolayer cultures, in reference to other in vitro models, are discussed. A new **Mycoplasma** agar medium (G-200 agar) with a defined tissue culture base and 10% horse serum is described.

L6 ANSWER 66 OF 87 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation. on

STN

ACCESSION NUMBER: 1980:128701 BIOSIS
DOCUMENT NUMBER: PREV198069003697; BA69:3697
TITLE: ADHERENCE OF ERYTHROCYTES TO **MYCOPLASMA**
-PNEUMONIAE.
AUTHOR(S): FELDNER J [Reprint author]; BREDT W; KAHANE I
CORPORATE SOURCE: ZENT HYG, INST ALLG HYG BAKTERIOL, UNIV FREIB, D-7800
FREIBURG IM BREISGAU, W GER
SOURCE: Infection and Immunity, (1979) Vol. 25, No. 1, pp. 60-67.
CODEN: INFIBR. ISSN: 0019-9567.
DOCUMENT TYPE: Article
FILE SEGMENT: BA
LANGUAGE: ENGLISH

AB The human pathogen *M. pneumoniae* adheres to a variety of cells, including erythrocytes. A hemadsorption technique was developed to quantitate adherence by photometric measurement of lysates of erythrocytes that attached to sheets of *M. pneumoniae* grown in cups of Linbro plates. Attachment of sheep erythrocytes (SE) increased with higher ionic strength, was unaffected by minor pH variations (6-9) and was blocked by anti-*M. pneumoniae* antiserum but was not inhibited by a variety of sugars, amino acids and bovine serum albumin. The reaction was time and temperature dependent. The temperature curve showed peaks at 14 and 28° C with untreated SE but only 1 peak at about 38° C with glutaraldehyde-treated SE. The temperature dependence indicated involvement of metabolic or membrane activities in the binding process. Trypsin treatment of the *M. pneumoniae* sheet abolished adherence of SE but was only partially effective with human erythrocytes and non-effective with rabbit erythrocytes. The binding capacity of the **mycoplasma** cells for SE was restored by incubation in growth medium for 3-4 h; this restoration was inhibited by 10 µg of chloramphenicol/ml. **Neuraminidase** treatment of SE removed their attachment capacity but had no effect on attachment of rabbit erythrocytes and only a slight effect on attachment of human erythrocytes. Pre-treatment of *M. pneumoniae* with neuraminic acid partially blocked the adherence of SE, whereas rabbit erythrocyte attachment was not affected. Attached SE could be detached by trypsin, but not by **neuraminidase**. For human and rabbit erythrocytes, the results suggest binding mechanisms other than the interaction between **neuraminidase**-sensitive receptors and protein-containing binding sites shown for SE.

L6 ANSWER 67 OF 87 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation. on
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ACCESSION NUMBER: 1979:129773 BIOSIS
DOCUMENT NUMBER: PREV197967009773; BA67:9773
TITLE: ADHERENCE OF **MYCOPLASMA**-GALLISEPTICUM TO HUMAN
ERYTHROCYTES.
AUTHOR(S): BANAI M [Reprint author]; KAHANE I; RAZIN S; BREDT W
CORPORATE SOURCE: BIOMEMBR RES LAB, DEP CLIN MICROBIOL, HEBR UNIV, HADASSAH
MED SCH, JERUSALEM, ISR
SOURCE: Infection and Immunity, (1978) Vol. 21, No. 2, pp. 365-372.
CODEN: INFIBR. ISSN: 0019-9567.
DOCUMENT TYPE: Article
FILE SEGMENT: BA
LANGUAGE: ENGLISH

AB Pathogenic **mycoplasmas** adhere to and colonize the epithelial lining of the respiratory and genital tracts of infected animals. An experimental system suitable for the quantitative study of **mycoplasma** adherence was developed. The system consists of human erythrocytes (RBC) and the avian pathogen **Mycoplasma gallisepticum**, in which membrane lipids were labeled. The amount of **mycoplasma** cells attached to the RBC, which was determined according to radioactivity measurements, decreased on increasing the pH or ionic strength of the attachment mixture. Attachment followed 1st-order kinetics and depended on temperature. The **mycoplasma** cell

population remaining in the supernatant fluid after exposure to RBC showed a much poorer ability to attach to RBC during a 2nd attachment test, indicating an unequal distribution of binding sites among cells within a given population. The gradual removal of sialic acid residues from the RBC by **neuraminidase** was accompanied by a decrease in **mycoplasma** attachment. Isolated glycophorin, the RBC membrane glycoprotein carrying almost all the sialic acid moieties of the RBC, inhibited *M. gallisepticum* attachment; asialoglycophorin and sialic acid itself were very poor inhibitors of attachment. Only part of the 125I-labeled glycophorin bound to **mycoplasmas** could be removed by **neuraminidase** or by exchange with unlabeled glycophorin. Glycophorin, representing the isolated major RBC receptor for *M. gallisepticum*, may bind to the **mycoplasmas** specifically, through its sialic acid moieties, and nonspecifically, through its exposed hydrophobic polypeptide moiety.

L6 ANSWER 68 OF 87 MEDLINE on STN DUPLICATE 21
 ACCESSION NUMBER: 79047249 MEDLINE
 DOCUMENT NUMBER: PubMed ID: 711320
 TITLE: Ciliated respiratory epithelial monolayers: new model for **Mycoplasma pneumoniae infection**.
 AUTHOR: Gabridge M G; Gunderson H; Schaeffer S L; Barden-Stahl Y D
 SOURCE: Infection and immunity, (1978 Jul) 21 (1) 333-6.
 Journal code: 0246127. ISSN: 0019-9567.
 PUB. COUNTRY: United States
 DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
 LANGUAGE: English
 FILE SEGMENT: Priority Journals
 ENTRY MONTH: 197901
 ENTRY DATE: Entered STN: 19900314
 Last Updated on STN: 19900314
 Entered Medline: 19790115

AB Hamster respiratory epithelial cells were cultured in a monolayer format, and 20% of the cells were ciliated. **Mycoplasma pneumoniae** attached to the epithelial cells in a **neuraminidase**-specific fashion and induced ciliostasis and cytonecrosis.

L6 ANSWER 69 OF 87 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation. on STN
 ACCESSION NUMBER: 1980:26141 BIOSIS
 DOCUMENT NUMBER: PREV198018026141; BR18:26141
 TITLE: PARTICIPATION OF BACTERIAL **NEURAMINIDASES** IN THE DEVELOPMENT OF **DISEASES**.
 AUTHOR(S): KOBRINSKII G D [Reprint author]
 CORPORATE SOURCE: RES LAB EXP IMMUNOBIOLOG, ACAD MED SCI USSR, MOSCOW, USSR
 SOURCE: Zhurnal Mikrobiologii Epidemiologii i Immunobiologii, (1978) No. 11, pp. 26-32.
 CODEN: ZMEIAV. ISSN: 0372-9311.
 DOCUMENT TYPE: Article
 FILE SEGMENT: BR
 LANGUAGE: RUSSIAN

L6 ANSWER 70 OF 87 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation. on STN
 ACCESSION NUMBER: 1977:228730 BIOSIS
 DOCUMENT NUMBER: PREV197764051094; BA64:51094
 TITLE: DIFFERENCES IN THE ATTACHMENT OF **MYCOPLASMA** -PNEUMONIAE CELLS AND MEMBRANES TO TRACHEAL EPITHELIUM.
 AUTHOR(S): GABRIDGE M G; BARDEN-STAHLE Y D; POLISKY R B; ENGELHARDT J A
 SOURCE: Infection and Immunity, (1977) Vol. 16, No. 3, pp. 766-772.
 CODEN: INFIBR. ISSN: 0019-9567.
 DOCUMENT TYPE: Article
 FILE SEGMENT: BA
 LANGUAGE: Unavailable

AB Hamster trachea organ cultures were exposed to isolated membranes of *M. pneumoniae*, PI 1428. Attachment, monitored by the uptake of tritiated membranes, was relatively insensitive to **neuraminidase** pretreatment, unlike the attachment of viable cells. Membrane attachment was optimal when explants were incubated with 50-100 µg of membrane protein/ml in minimal essential medium broth while gently being rotated (1 rpm) in a roller apparatus for 90-120 min at 37° C. Saturation of the receptor sites with viable cells failed to inhibit subsequent membrane attachment. Induction of squamous metaplasia by extended cultivation of tracheal explants in a vitamin A-free medium reduced the content of ciliated cells without significantly affecting total cell viability, but did not alter the attachment of *M. pneumoniae* membranes. Collectively, the data indicate that the mechanism of attachment of *M. pneumoniae* membranes to respiratory epithelium is distinct from the receptor site-mediated attachment of *M. pneumoniae* cells.

L6 ANSWER 71 OF 87 MEDLINE on STN DUPLICATE 22

ACCESSION NUMBER: 77140084 MEDLINE

DOCUMENT NUMBER: PubMed ID: 557458

TITLE: Effect of squamous metaplasia on **infection** of hamster trachea organ cultures with **Mycoplasma pneumoniae**.

AUTHOR: Engelhardt J A; Gabridge M G

SOURCE: Infection and immunity, (1977 Feb) 15 (2) 647-55.

Journal code: 0246127. ISSN: 0019-9567.

PUB. COUNTRY: United States

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English

FILE SEGMENT: Priority Journals

ENTRY MONTH: 197705

ENTRY DATE: Entered STN: 19900313

Last Updated on STN: 19900313

Entered Medline: 19770525

AB An organ culture system for hamster trachea was developed for maintenance of the ciliated respiratory epithelium during periods of extended cultivation (i.e., greater than 20 days). Evaluation of five serum types showed that horse serum and fetal calf serum were best for the maintenance of epithelial ciliary activity and morphology. Rings that were opened on one side ("split rings") had the best maintenance of the ciliated epithelium as judged by the retention of ciliary activity and normal histological appearance after 3 to 4 weeks in culture. The in vitro induction of squamous metaplasia was achieved by cultivating explants in Waymouth MAB 87/3 (vitamin A-free) medium, without serum. This system allowed a direct comparison of the effects of **Mycoplasma pneumoniae infection** in two epithelial types, ciliated pseudostratified columnar and keratinizing squamous. Attachment of ¹⁴C-labeled **mycoplasmas** was more than twofold greater in the normal epithelium. Pretreatment of explants with **neuraminidase** decreased attachment for both squamous and pseudostratified epithelial surfaces to a similar basal level. Recovery of viable organisms from infected tissue of both epithelial types indicated that the organism titer remained essentially constant during the **infection** period, but was significantly higher for the pseudostratified ciliated epithelium. These results suggest that specific receptor sites for *M. pneumoniae* are markedly reduced by the induction of squamous metaplasia and, hence, appear to be specific for the normal respiratory surface containing goblet cells and pseudostratified, ciliated epithelial cells.

L6 ANSWER 72 OF 87 EMBASE COPYRIGHT 2005 ELSEVIER INC. ALL RIGHTS RESERVED. on STN

ACCESSION NUMBER: 78045969 EMBASE

DOCUMENT NUMBER: 1978045969

TITLE: Effect of squamous metaplasia on **infection** of hamster trachea organ cultures with **Mycoplasma**

pneumoniae.
 AUTHOR: Engelhardt J.A.; Gabridge M.G.
 CORPORATE SOURCE: Dept. Microbiol., Sch. Bas. Med. Sci., Univ. Illinois,
 Urbana, Ill. 61801, United States
 SOURCE: Infection and Immunity, (1977) 5/2 (647-655).
 CODEN: INFIBR
 DOCUMENT TYPE: Journal
 FILE SEGMENT: 004 Microbiology
 016 Cancer
 023 Nuclear Medicine
 005 General Pathology and Pathological Anatomy
 LANGUAGE: English

AB An organ culture system for hamster trachea was developed for maintenance of the ciliated respiratory epithelium during periods of extended cultivation (i.e., >20 days). Evaluation of 5 serum types showed that horse serum and fetal calf serum were best for the maintenance of epithelial ciliary activity and morphology. Rings that were opened on one side ('split rings') had the best maintenance of the ciliated epithelium as judged by the retention of ciliary activity and normal histologic appearance after 3 to 4 wk in culture. The in vitro induction of squamous metaplasia was achieved by cultivating explants in Waymouth MAB 87/3 (vitamin A free) medium, without serum. This system allowed a direct comparison of the effects of **Mycoplasma pneumoniae infection** in two epithelial types, ciliated pseudostratified columnar and keratinizing squamous. Attachment of ¹⁴C labeled **mycoplasmas** was more than twofold greater in the normal epithelium. Pretreatment of explants with **neuraminidase** decreased attachment for both squamous and pseudostratified epithelial surfaces to a similar basal level. Recovery of viable organisms from infected tissue of both epithelial types indicated that the organism titer remained essentially constant during the **infection** period, but was significantly higher for the pseudostratified ciliated epithelium. These results suggest that specific receptor sites for *M. pneumoniae* are markedly reduced by the induction of squamous metaplasia and, hence, appear to be specific for the normal respiratory surface containing goblet cells and pseudostratified, ciliated epithelial cells.

L6 ANSWER 73 OF 87 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation. on STN

ACCESSION NUMBER: 1976:172780 BIOSIS
 DOCUMENT NUMBER: PREV197662002780; BA62:2780
 TITLE: ATTACHMENT OF **MYCOPLASMA**-PNEUMONIAE TO
 RESPIRATORY EPITHELIUM.
 AUTHOR(S): POWELL D A; HU P C; WILSON M; COLLIER A M; BASEMAN J B
 SOURCE: Infection and Immunity, (1976) Vol. 13, No. 3, pp. 959-966.
 CODEN: INFIBR. ISSN: 0019-9567.
 DOCUMENT TYPE: Article
 FILE SEGMENT: BA
 LANGUAGE: Unavailable

AB The attachment of radioisotope-labeled *M. pneumoniae* to hamster tracheal rings in organ culture was examined by radioautography and liquid scintillation counting. Radioautographs of individual rings exposed for 8 h to [³H]thymidine-labeled virulent *M. pneumoniae* revealed a dense extracellular collection of emulsion grains along the luminal surface of epithelial cells. Similar exposure of rings to isotope-labeled avirulent *M. pneumoniae* resulted in no accumulation of emulsion grains. The numbers of attached virulent **mycoplasmas**, as measured by liquid scintillation counting of infected rings, increased in a nearly linear fashion over an 8-h incubation period. Viability of the **mycoplasmas** and metabolic integrity of the tracheal rings were important for optimal attachment. Pretreatment of rings with **neuraminidase** or Na periodate significantly impaired organism adherence. A specificity of interaction between virulent *M. pneumoniae* and tracheal epithelial cells that can be further examined through the use

of isotopically labeled **mycoplasmas** is suggested.

L6 ANSWER 74 OF 87 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation. on STN

ACCESSION NUMBER: 1977:222761 BIOSIS
DOCUMENT NUMBER: PREV197764045125; BA64:45125
TITLE: NEWCASTLE **DISEASE** VIRUS O AGGLUTININS IN RELATION TO T AGGLUTININS IN LABORATORY ANIMALS AND IN HUMAN PATIENTS INFECTED WITH **MYCOPLASMA**-PNEUMONIAE.
AUTHOR(S): PYHALA R
SOURCE: Annales Zoologici Fennici, (1976) Vol. 13, No. 3, pp. 139-147.
CODEN: AZOFAO. ISSN: 0003-455X.
DOCUMENT TYPE: Article
FILE SEGMENT: BA
LANGUAGE: Unavailable
AB A serological relationship exists between the agglutinins detected with erythrocytes modified by the B1 strain of Newcastle **disease** virus (NDV-O agglutinins) and the T agglutinins detected with the RDE[receptor destroying enzyme(Vibrio cholerae **neuraminidase**)]-O test system. This relationship was studied with sera of unimmunized adult laboratory animals (rabbit, guinea pig, sheep, mouse, rat) and of human patients infected with **Mycoplasma pneumoniae**. In laboratory animals all moieties of the B1-O agglutinins were usually demonstrable with the RDE-O test system; in contrast, the RDE-O agglutinins frequently included a component which was not demonstrable with the B1-O test system. This component was not found in human patients. At different temperatures the B1-O agglutinins produced by the patients reacted in the same fashion as the natural B1-O and RDE-O agglutinins of the animals.

L6 ANSWER 75 OF 87 MEDLINE on STN

ACCESSION NUMBER: 77041007 MEDLINE
DOCUMENT NUMBER: PubMed ID: 790846
TITLE: [Pathogenicity factors in **mycoplasma** and their possible significance in mixed **infections** with bacteria and Candida albicans (proceedings)]. Pathogenitätsfaktoren von Mykoplasmen und deren mögliche Bedeutung in Mischinfektionen mit Bakterien und Candida albicans.
AUTHOR: Goeth H; Appel K R; Hoxer K
SOURCE: Zentralblatt für Bakteriologie, Parasitenkunde, Infektionskrankheiten und Hygiene. Erste Abteilung Originale. Reihe A: Medizinische Mikrobiologie und Parasitologie, (1976 Aug) 235 (1-3) 134-41.
Journal code: 0331570. ISSN: 0300-9688.
PUB. COUNTRY: GERMANY, WEST: Germany, Federal Republic of
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: German
FILE SEGMENT: Priority Journals
ENTRY MONTH: 197612
ENTRY DATE: Entered STN: 19900313
Last Updated on STN: 19980206
Entered Medline: 19761230

L6 ANSWER 76 OF 87 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation. on STN

ACCESSION NUMBER: 1975:231944 BIOSIS
DOCUMENT NUMBER: PREV197560061940; BA60:61940
TITLE: MICHAELIS CONSTANTS OF **NEURAMINIDASES** OF PATHOGENIC AND APATHOGENIC MICROORGANISMS.
AUTHOR(S): MUELLER H E; VON NICOLAI H; ZILLIKEN F
SOURCE: Zeitschrift für Naturforschung Teil C Biochemie Biophysik Biologie Virologie, (1975) Vol. 30, No. 3, pp. 417-419.
CODEN: ZNFCAP. ISSN: 0341-0471.

DOCUMENT TYPE: Article
FILE SEGMENT: BA
LANGUAGE: Unavailable

L6 ANSWER 77 OF 87 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation. on
STN

ACCESSION NUMBER: 1975:231886 BIOSIS
DOCUMENT NUMBER: PREV197560061882; BA60:61882
TITLE: GROWTH AND CYTO PATHOLOGY OF **MYCOPLASMA**-SYNOVIAE
IN CHICKEN EMBRYO CELL CULTURES.
AUTHOR(S): ALDRIDGE K E
SOURCE: Infection and Immunity, (1975) Vol. 12, No. 1, pp. 198-204.
CODEN: INFIBR. ISSN: 0019-9567.
DOCUMENT TYPE: Article
FILE SEGMENT: BA
LANGUAGE: Unavailable

L6 ANSWER 78 OF 87 MEDLINE on STN

ACCESSION NUMBER: 75121347 MEDLINE
DOCUMENT NUMBER: PubMed ID: 1118664
TITLE: Haemagglutination and haemagglutination inhibition with
Mycoplasma synoviae.
AUTHOR: Windsor G D; Thompson G W; Baker N W
SOURCE: Research in veterinary science, (1975 Jan) 18 (1) 59-63.
Journal code: 0401300. ISSN: 0034-5288.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 197506
ENTRY DATE: Entered STN: 19900310
Last Updated on STN: 19900310
Entered Medline: 19750606

AB A haemagglutinating antigen prepared from cultures of M synoviae WVU 1853
successfully detected homologous haemagglutination inhibition (HI) in sera
of fowls and turkeys inoculated with M synoviae. Nonspecific HI was
encountered with normal fowl sera but this was removed by treatment with
receptor destroying enzyme. It is suggested that M synoviae. HA antigen
will be a useful reagent for the diagnosis of M synoviae **infection**

L6 ANSWER 79 OF 87 MEDLINE on STN

DUPLICATE 23

ACCESSION NUMBER: 76007091 MEDLINE
DOCUMENT NUMBER: PubMed ID: 1159572
TITLE: Histochemical identification of glycoproteins in pig
bronchial epithelium: (a) normal and (b) hypertrophied from
enzootic pneumonia.
AUTHOR: Jones R; Baskerville A; Reid L
SOURCE: Journal of pathology, (1975 May) 116 (1) 1-11.
Journal code: 0204634. ISSN: 0022-3417.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 197512
ENTRY DATE: Entered STN: 19900313
Last Updated on STN: 19900313
Entered Medline: 19751204

AB The glycoproteins in the normal pig bronchial gland are identified by the
combined Alcian Blue (AB)-periodic acid Schiff (PAS) technique, with the
use of **sialidase** digestion and AB staining either at pH 2-6 or
at pH 1-0. In enzootic pneumonia (produced experimentally by
infection with **Mycoplasma** hyorhinis) the bronchial gland
hypertrophies, mucous and serous cells both increase, in number and size;

hence the total glycoprotein content of the gland increases. The distribution of glycoproteins in the hypertrophied gland differs from that in the normal. Quantitative analysis of the mucous cells shows that in the hypertrophied gland the acid glycoprotein is increased relative to the neutral. There is also a relative change in the amounts of **sialidase**-sensitive sialomucin and sulphomucin; both are significantly increased at the expense of the **sialidase**-resistant sialomucin. Qualitative analysis of the serous cells shows that in the normal gland most of the glycoprotein is neutral and that the small amount of acid glycoprotein is **sialidase**-resistant sialomucin. In the hypertrophied gland there is relatively more acid glycoprotein which is either **sialidase**-resistant sialomucin or sulphomucin; in addition, in pigs with enzootic pneumonia there is an increase in the height of the bronchial epithelium and a depletion in both goblet cell number and glycoprotein content, which latter has more neutral glycoprotein and less acid glycoprotein.

L6 ANSWER 80 OF 87 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation. on STN

ACCESSION NUMBER: 1972:189870 BIOSIS
DOCUMENT NUMBER: PREV197254019864; BA54:19864
TITLE: STUDIES ON ATTACHMENT AND INGESTION PHASES OF PHAGOCYTOSIS OF **MYCOPLASMA**-PULMONIS BY MOUSE PERITONEAL MACROPHAGES.
AUTHOR(S): JONES T C; YEH S; HIRSCH J G
SOURCE: Proceedings of the Society for Experimental Biology and Medicine, (1972) Vol. 139, No. 2, pp. 464-470.
CODEN: PSEBAA. ISSN: 0037-9727.
DOCUMENT TYPE: Article
FILE SEGMENT: BA
LANGUAGE: Unavailable

L6 ANSWER 81 OF 87 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation. on STN

ACCESSION NUMBER: 1972:161266 BIOSIS
DOCUMENT NUMBER: PREV197253061266; BA53:61266
TITLE: **NEURAMINIDASE** ACTIVITY IN **MYCOPLASMA** -GALLISEPTICUM.
AUTHOR(S): SETHI K K; MULLER H E
SOURCE: Infection and Immunity, (1972) Vol. 5, No. 2, pp. 260-262.
CODEN: INFIBR. ISSN: 0019-9567.
DOCUMENT TYPE: Article
FILE SEGMENT: BA
LANGUAGE: Unavailable

L6 ANSWER 82 OF 87 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation. on STN

ACCESSION NUMBER: 1972:84102 BIOSIS
DOCUMENT NUMBER: PREV197208084102; BR08:84102
TITLE: FACTORS INFLUENCING PATHOGENICITY OF AVIAN **MYCOPLASMOSIS**.
AUTHOR(S): GERLACH H
SOURCE: Medical Microbiology and Immunology, (1972) Vol. 157, No. 2, pp. 179.
CODEN: MMIYAO. ISSN: 0300-8584.
DOCUMENT TYPE: Article
FILE SEGMENT: BR
LANGUAGE: Unavailable

L6 ANSWER 83 OF 87 MEDLINE on STN DUPLICATE 24

ACCESSION NUMBER: 72086068 MEDLINE
DOCUMENT NUMBER: PubMed ID: 5167481
TITLE: Growth and pathogenicity studies of **Mycoplasma** gallisepticum in chicken tracheal organ cultures.
AUTHOR: Cherry J D; Taylor-Robinson D

SOURCE: Journal of medical microbiology, (1971 Nov) 4 (4) 441-9.
Journal code: 0224131. ISSN: 0022-2615.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 197203
ENTRY DATE: Entered STN: 19900310
Last Updated on STN: 19900310
Entered Medline: 19720323

L6 ANSWER 84 OF 87 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation. on
STN DUPLICATE 25

ACCESSION NUMBER: 1971:104979 BIOSIS
DOCUMENT NUMBER: PREV197152014979; BA52:14979
TITLE: GROWTH AND PATHOGENESIS OF **MYCOPLASMA**
-MYCOIDES-VAR-CAPRI IN CHICKEN EMBRYO TRACHEAL ORGAN
CULTURES.
AUTHOR(S): CHERRY J D; TAYLOR-ROBINSON D
SOURCE: Infection and Immunity, (1970) Vol. 2, No. 4, pp. 431-438.
CODEN: INFIBR. ISSN: 0019-9567.
DOCUMENT TYPE: Article
FILE SEGMENT: BA
LANGUAGE: Unavailable

L6 ANSWER 85 OF 87 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation. on
STN

ACCESSION NUMBER: 1969:178112 BIOSIS
DOCUMENT NUMBER: PREV196950116112; BA50:116112
TITLE: UTILIZATION OF NEURAMINIC-ACID RECEPTORS BY
MYCOPLASMAS MYCOPLASMATA ENZ **NEURAMINIDASE**
MYCOPLASMA-PNEUMONIAE HUMAN FOWL ERYTHROCYTES
MYCOPLASMA-GALLISEPTICUM NEOPL HELA CELLS
MYCOPLASMA-SYNOVIAE **MYCOPLASMA**-WR1.
AUTHOR(S): MANCHEE R J; TAYLOR-ROBINSON D
SOURCE: Journal of Bacteriology, (1969) Vol. 98, No. 3, pp.
914-919.
CODEN: JOBAAY. ISSN: 0021-9193.
DOCUMENT TYPE: Article
FILE SEGMENT: BA
LANGUAGE: Unavailable

L6 ANSWER 86 OF 87 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation. on
STN

ACCESSION NUMBER: 1969:195037 BIOSIS
DOCUMENT NUMBER: PREV196950133037; BA50:133037
TITLE: STUDIES ON THE NATURE OF RECEPTORS INVOLVED IN ATTACHMENT
OF TISSUE CULTURE CELLS TO **MYCOPLASMAS**
MYCOPLASMA-GALLISEPTICUM **MYCOPLASMA**
-PNEUMONIAE **MYCOPLASMA**-HOMINIS **MYCOPLASMA**
-SALIVARIUM ENZ **NEURAMINIDASE** NEOPL HELA.
AUTHOR(S): MANCHEE R J; TAYLOR-ROBINSON D
SOURCE: British Journal of Experimental Pathology, (1969) Vol. 50,
No. 1, pp. 66-75.
CODEN: BJEPAS. ISSN: 0007-1021.
DOCUMENT TYPE: Article
FILE SEGMENT: BA
LANGUAGE: Unavailable

L6 ANSWER 87 OF 87 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation. on
STN

ACCESSION NUMBER: 1969:200674 BIOSIS
DOCUMENT NUMBER: PREV196950003664; BA50:3664
TITLE: ABSORPTION OF **MYCOPLASMA**-PNEUMONIAE TO

NEURAMINIC-ACID RECEPTORS OF VARIOUS CELLS AND POSSIBLE
 ROLE IN VIRULENCE MONKEY RAT GUINEA-PIG CHICKEN
 ERYTHROCYTES EPITHELIAL CELLS ENZ **NEURAMINIDASE**
 INFLUENZA B VIRUS **MYCOPLASMA**-GALLISEPTICUM
MYCOPLASMA-PULMONIS **MYCOPLASMA**-ORALE.

AUTHOR(S): SOBESLAVSKY O; PRESCOTT B; CHANOCK R M
 SOURCE: Journal of Bacteriology, (1968) Vol. 96, No. 3, pp.
 695-705.
 CODEN: JOBAAY. ISSN: 0021-9193.
 DOCUMENT TYPE: Article
 FILE SEGMENT: BA
 LANGUAGE: Unavailable

=> d his

(FILE 'HOME' ENTERED AT 14:05:18 ON 10 JAN 2005)

FILE 'MEDLINE, EMBASE, BIOSIS, BIOTECHDS, SCISEARCH, HCAPLUS, NTIS,
 LIFESCI' ENTERED AT 14:05:42 ON 10 JAN 2005

L1 73150 S MYCOPLASMA
 L2 63554 S NEURAMINIDASE? OR SIALIDASE?
 L3 224 S L1 AND L2
 L4 139 S (INFECTION? OR DISEASE?) AND L3
 L5 2620647 S CANCER OR "HODGKIN'S" AND L4
 L6 87 DUP REM L4 (52 DUPLICATES REMOVED)
 L7 652 S (CANCER OR "HODGKIN'S") AND L6

=> e higuchi m/au

E1 7 HIGUCHI L M/AU
 E2 1 HIGUCHI LESLIE M/AU
 E3 3027 --> HIGUCHI M/AU
 E4 45 HIGUCHI M A/AU
 E5 53 HIGUCHI M D/AU
 E6 37 HIGUCHI M D L/AU
 E7 32 HIGUCHI M DE L/AU
 E8 2 HIGUCHI M DE LOURDES/AU
 E9 1 HIGUCHI M GREATER THAN OR EQUAL TO/AU
 E10 136 HIGUCHI M L/AU
 E11 1 HIGUCHI M L H M D/AU
 E12 1 HIGUCHI MAHO/AU

=> s e3

L8 3027 "HIGUCHI M"/AU

=> e schenkman s/au

E1 14 SCHENKMAN ROCILDA P F/AU
 E2 1 SCHENKMAN ROCILDA PERAZZINI FUKASAWA/AU
 E3 344 --> SCHENKMAN S/AU
 E4 4 SCHENKMAN S */AU
 E5 1 SCHENKMAN S P/AU
 E6 5 SCHENKMAN S S/AU
 E7 2 SCHENKMAN SELVA S/AU
 E8 106 SCHENKMAN SERGIO/AU
 E9 1 SCHENKMAN SIMONE/AU
 E10 1 SCHENKMANM RONNI L/AU
 E11 2 SCHENKMANN A/AU
 E12 3 SCHENKMANN N S/AU

=> s e3

L9 344 "SCHENKMAN S"/AU

=> s 18 or 19

L10 3371 L8 OR L9

=> d his

(FILE 'HOME' ENTERED AT 14:05:18 ON 10 JAN 2005)

FILE 'MEDLINE, EMBASE, BIOSIS, BIOTECHDS, SCISEARCH, HCAPLUS, NTIS, LIFESCI' ENTERED AT 14:05:42 ON 10 JAN 2005

L1 73150 S MYCOPLASMA
L2 63554 S NEURAMINIDASE? OR SIALIDASE?
L3 224 S L1 AND L2
L4 139 S (INFECTION? OR DISEASE?) AND L3
L5 2620647 S CANCER OR "HODGKIN'S" AND L4
L6 87 DUP REM L4 (52 DUPLICATES REMOVED)
L7 652 S (CANCER OR "HODGKIN'S") AND L6
E HIGUCHI M/AU
L8 3027 S E3
E SCHENKMAN S/AU
L9 344 S E3
L10 3371 S L8 OR L9

=> s l6 and l10

L11 2 L6 AND L10

=> d 1-2 ibib ab

L11 ANSWER 1 OF 2 BIOTECHDS COPYRIGHT 2005 THE THOMSON CORP. on STN

ACCESSION NUMBER: 2004-00309 BIOTECHDS

TITLE: Use of an agent that prevents or inhibits **Mycoplasma infection**, for manufacturing a medicament for treating or preventing a disorder associated with increased cell proliferation, e.g. atherosclerotic vascular **disease** or malignancy;
recombinant Trypanosoma cruzi protein application in **infection**, tumor and vascular **disease** therapy

AUTHOR: HIGUCHI M D L; **SCHENKMAN S**

PATENT ASSIGNEE: HIGUCHI M D L; **SCHENKMAN S**

PATENT INFO: US 2003124109 3 Jul 2003

APPLICATION INFO: US 2002-86913 1 Mar 2002

PRIORITY INFO: BR 2001-2648 3 Jul 2001; BR 2000-2989 3 Jul 2000

DOCUMENT TYPE: Patent

LANGUAGE: English

OTHER SOURCE: WPI: 2003-810968 [76]

AB DERWENT ABSTRACT:

NOVELTY - Use of an agent that prevents or inhibits **Mycoplasma infection** for manufacturing a medicament for treating a disorder associated with increased cell proliferation.

DETAILED DESCRIPTION - An INDEPENDENT CLAIM is included for a composition for treating or preventing **Mycoplasma infection** in a subject suffering from a disorder associated with increased cell proliferation or a co-**infection** with **mycoplasma** and a second microbe, comprising an agent that prevents or inhibits sialic acid-mediated attachment of **mycoplasma** to cells of the subject.

BIOTECHNOLOGY - Preferred Composition: The agent is an antibiotic or an enzyme having an activity consisting of **neuraminidase** and/or trans-**sialidase** activity. The enzyme is derived from a Trypanosoma cruzi microorganism, where the enzyme is a native or a recombinant enzyme. The enzyme has a fully defined sequence of 669 amino acids given in the specification. A vector containing the DNA insert having a fully defined sequence of 2010 bp given in the specification produces the enzyme.

ACTIVITY - Antibacterial; Antiarteriosclerotic; Cytostatic; Anti-HIV. A 64-year-old female patient with a palpable abdominal mass and

a tumoral mass in the rectum was administered 50 ml of native trans-**sialidase** (TSN) intraperitoneally on alternate days for a period of 14 days. On day 23, with **mycoplasmas** confirmed in the bone marrow, erythromycin (500 mg/day) was given for a further 20 days. Clinical improvement and normalization of blood leukocytes was seen after 2 days. Considering the important clinical improvement and reduction in abdominal mass, a second session of TSN was administered. The patient demonstrated improvement in general clinical status. Tomography detected a reduction in tumoral mass. Results showed that trans-**sialidase** is effective as a drug in the treatment of neoplasia, removing **mycoplasmas** from the neoplastic cells leading to their apoptosis.

MECHANISM OF ACTION - **Neuraminidase**; Trans-**sialidase**.

USE - The composition or the agent that prevents or inhibits **mycoplasma infection** is useful for manufacturing a medicament for treating or preventing a disorder associated with increased cell proliferation, e.g. atherosclerotic vascular **disease** or malignant **disease**, or a **disease** associated with co-**infection** with **mycoplasma** and a second microbe such as human immunodeficiency virus or a Chlamydia microbe (all claimed).

ADMINISTRATION - The amount of the enzyme administered is about 106-1013 units per day. Administration may be intravenous, intraperitoneal, intrathecal, oral, by inhalation, subcutaneous, or intramuscular. (32 pages)

L11 ANSWER 2 OF 2 BIOTECHDS COPYRIGHT 2005 THE THOMSON CORP. on STN

ACCESSION NUMBER: 2002-08674 BIOTECHDS

TITLE: Composition useful for treatment of **mycoplasma infection** and **diseases** associated with cell proliferation e.g. malignancy or with co-**infection** with another microbe, comprises agent inhibiting sialic acid-mediated attachment of **mycoplasma**; native or recombinant enzyme treatment and vector-mediated gene transfer and expression in host cell for **disease** therapy or prevention

AUTHOR: HIGUCHI M D L; **SCHENKMAN S**

PATENT ASSIGNEE: HIGUCHI M D L; **SCHENKMAN S**

PATENT INFO: WO 2002002050 10 Jan 2002

APPLICATION INFO: WO 2000-BR83 3 Jul 2000

PRIORITY INFO: BR 2000-2989 3 Jul 2000

DOCUMENT TYPE: Patent

LANGUAGE: English

OTHER SOURCE: WPI: 2002-154675 [20]

AB DERWENT ABSTRACT:

NOVELTY - A composition useful for treating or preventing **mycoplasma infection** in a subject suffering from a disorder characterized by increased cell proliferation or by co-**infection** with a second microbe comprising an agent that prevents or inhibits sialic acid-mediated attachment of **mycoplasma** to the subject's cells, is new.

DETAILED DESCRIPTION - An INDEPENDENT CLAIM is also included for use of an agent preventing/inhibiting **mycoplasma infection** in medicaments to treat disorders characterized by increase cell proliferation.

BIOTECHNOLOGY - Preferred Composition: The agent is preferably an enzyme (native or recombinant) with **neuraminidase** and/or trans-**sialidase** activity, especially derived from *Trypanosoma cruzi*. It preferably has fully defined sequence (I) of 669 amino acids as given in the specification. The medicament preferably includes a vector comprising DNA insert of a fully defined sequence (II) of 2010 base pairs as given in the specification, producing the preferred enzyme having sequence (I) as above.

ACTIVITY - Antiatherosclerotic; antibacterial; antiviral; anti-HIV;

cytostatic; vasotropic. A laboratory rat population was determined to be infected with both **Mycoplasma pulmonis** and **Chlamydia pneumoniae** using standard immunohistological techniques and histological examination of various organs. Nine adult rats (approximately 285 g; seven males, two females) presenting conjunctivitis, slow movements and, in one rat severe otitis and another severe weight loss, were allocated to groups A or B. A (Control group) comprised three animals, two of which were killed without injecting any substance and one receiving an inactivated form of 'TSC' (recombinantly produced catalytic portion of *Trypanosoma cruzi* trans-sialidase) for 5 consecutive days. B comprised five animals receiving 'TNS' (complete, native *Trypanosoma cruzi* trans-sialidase) (0.5 ml/animal, every 2 days) and sacrificed after 7, 9, and 12 days, and one rat receiving active TSC (140 microgram/day, five consecutive days) and killed after 7 days. Group B rats showed a clear improvement in symptoms, becoming more agile, requiring more ether to anesthetize them and becoming more difficult to restrain. The animal with otitis showed less equilibrium loss and the animal with severe weight loss gained weight. Since the lung was the most frequently injured organ, pulmonary alterations were examined using electron microscopy, confocal laser microscopy and immunohistochemistry. Histological sections showed that treated animals presented resolving pneumonitis after 7 d. After 9-12 days *M. pulmonis* were almost absent from alveoli and mean *C. pneumoniae* positive cell numbers in alveoli had decreased, compatible with regression of *C. pneumoniae* infection. Results are given in the specification.

MECHANISM OF ACTION - Inhibits sialic acid mediated attachment of **mycoplasma** to cells.

USE - The compositions are useful to treat **diseases** associated with undesirable cell proliferation, such as atherosclerotic vascular **disease** and malignancy (both claimed), by reducing or preventing **mycoplasma** infection. They also useful to treat **diseases** associated with infection with other infectious organisms co-occurring with **mycoplasma** (and typically increasing the virulence of both pathogens), especially human immunodeficiency virus or chlamydia species. They can be used to treat such **diseases** in humans or other animals, and can be administered in conjunction with conventional agents e.g. anti-platelet or chemotherapeutic agents. (63 pages)

=> d his

(FILE 'HOME' ENTERED AT 14:05:18 ON 10 JAN 2005)

FILE 'MEDLINE, EMBASE, BIOSIS, BIOTECHDS, SCISEARCH, HCAPLUS, NTIS, LIFESCI' ENTERED AT 14:05:42 ON 10 JAN 2005

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L1      73150 S MYCOPLASMA
L2      63554 S NEURAMINIDASE? OR SIALIDASE?
L3      224 S L1 AND L2
L4      139 S (INFECTION? OR DISEASE?) AND L3
L5      2620647 S CANCER OR "HODGKIN'S" AND L4
L6      87 DUP REM L4 (52 DUPLICATES REMOVED)
L7      652 S (CANCER OR "HODGKIN'S") AND L6
        E HIGUCHI M/AU
L8      3027 S E3
        E SCHENKMAN S/AU
L9      344 S E3
L10     3371 S L8 OR L9
L11     2 S L6 AND L10
```

=> s l6 and (prevent or inhibit)

```
L12     8 L6 AND (PREVENT OR INHIBIT)
```

=> d 1-8 ibib ab

L12 ANSWER 1 OF 8 MEDLINE on STN
 ACCESSION NUMBER: 2004419768 IN-PROCESS
 DOCUMENT NUMBER: PubMed ID: 15325005
 TITLE: Trypanosoma cruzi trans-**sialidase** as a new
 therapeutic tool in the treatment of chronic inflammatory
diseases: possible action against
mycoplasma and chlamydia.
 AUTHOR: de Lourdes Higuchi Maria
 CORPORATE SOURCE: Pathology Laboratory, Heart Institute (InCor) of Clinical
 Hospital, School of Medicine of Sao Paulo University, Av.
 Dr Eneas de Carvalho Aguiar 44, 05403-000 Sao Paulo, SP,
 Brazil.. anplourdes@incor.usp.br
 SOURCE: Medical hypotheses, (2004) 63 (4) 616-23.
 Journal code: 7505668. ISSN: 0306-9877.
 PUB. COUNTRY: Scotland: United Kingdom
 DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
 LANGUAGE: English
 FILE SEGMENT: IN-PROCESS; NONINDEXED; Priority Journals
 ENTRY DATE: Entered STN: 20040825
 Last Updated on STN: 20041219

AB The present paper proposes a new therapy using Trypanosoma cruzi trans-**sialidase** to treat **diseases** with unclear pathogenesis that present in common chronic inflammation and fibrosis. This hypothesis is based on recent findings that co-**infection** with **mycoplasma** and chlamydia is present in many of these **diseases** and that this enzyme was capable to eliminate or decrease the co-**infection** from the host. We identified that **mycoplasmas** and chlamydias are present in atherosclerosis, aortic valve stenosis, dilated cardiomyopathy, chronic chagasic myocarditis and cancer. We hypothesized that mycoplasmal **infection** may induce immunodepression in the host, favoring proliferation of pre-existent chlamydial **infection** and that elimination of **mycoplasma** would lead to improvement of the immune system resistance and the control of chlamydial proliferation. **Mycoplasma** has a particular parasitic relationship with host cells, involving strong adherence of their membranes, making it extremely difficult to eradicate mycoplasmal **infection** from the host. A new therapeutic approach is suggested using one or more agents that **prevent** or **inhibit** the adherence of **mycoplasma** to host cell membranes by removing sialic acid residues and preventing oxidation of the cells. The use of a **neuraminidase** enzyme, particularly the T. cruzi trans-**sialidase** enzyme, associated with treatment using anti-oxidating agents is proposed. Preliminary experimental animal and laboratory tests showed good results. The proposal that trans-**sialidase** from T. cruzi is efficient in combating co-**infection** of **mycoplasma** and chlamydia is based, at least in part, on the observation that chagasic patients suffering from T. cruzi **infection** present less **mycoplasma** and chlamydia **infection** in their tissues. Also, a lower incidence of the **diseases** above described to be related to **mycoplasma** **infection** is observed in chagasic patients. It is also hypothesized that co-**infection** with **mycoplasma** and chlamydia may induce oxidation of the host cells. Anti-oxidants such as those present in plant extracts may also be used in the treatment. Other **diseases** such as chronic hepatitis, glomerulonephritis, Multiple Sclerosis, Alzheimer's Syndrome and idiopathic encephalitis are other examples of chronic **diseases** where **mycoplasma** and chlamydia might be present, as they have the characteristics of unknown etiology, persistent chronic inflammation and fibrosis.
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L12 ANSWER 2 OF 8 EMBASE COPYRIGHT 2005 ELSEVIER INC. ALL RIGHTS RESERVED.
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ACCESSION NUMBER: 2002046997 EMBASE
 TITLE: Infectious **diseases**.
 AUTHOR: Erard Ph.
 CORPORATE SOURCE: Dr. Ph. Erard, Departement de Medecine, Hopital des
 Cadolles, 2000 Neuchatel, Switzerland. ph.erard@net2000.ch
 SOURCE: Medecine et Hygiene, (16 Jan 2002) 60/2375 (111-114).
 Refs: 34
 ISSN: 0025-6749 CODEN: MEHGAB
 COUNTRY: Switzerland
 DOCUMENT TYPE: Journal; Article
 FILE SEGMENT: 004 Microbiology
 015 Chest Diseases, Thoracic Surgery and Tuberculosis
 037 Drug Literature Index
 LANGUAGE: English
 SUMMARY LANGUAGE: English

AB About 75% of antibiotic prescriptions in the outpatient setting are made for upper respiratory tract **infections**. New guidelines have been issued this year emphasizing that the vast majority of antibiotic prescriptions are not justified. More importantly, these unnecessary prescriptions are likely to contribute considerably to the emergence of antibiotic resistance. Community-acquired pneumonia is mainly caused by pneumococci and **mycoplasma**. Empirical treatment should therefore cover both groups of pathogens. Several studies have shown that **neuraminidase**-inhibitors, when administered prophylactically to family members of an index case with influenza, can **prevent** intrafamilial transmission of influenza. While a single dose of prophylactic doxycycline given shortly after a tick bite and removal of tick, can **prevent** the transmission of the Lyme agent in areas with a high (>3%) transmission rate, antibiotic treatment of patients with chronic fatigue having suffered of Lyme **disease** was of no benefit. Self-treatment of young women with acute uncomplicated cystitis has been used in clinical practice for many years. A recent prospective study validates this approach. These and other new studies should hopefully contribute to a rational and economic usage of antibiotics.

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ACCESSION NUMBER: 80142806 EMBASE
 DOCUMENT NUMBER: 1980142806
 TITLE: **Mycoplasmas** and ureaplasmas in infertility and abortion.
 AUTHOR: Friberg J.
 CORPORATE SOURCE: Dept. Obstet. Gynecol., Downstate Med. Cent., Brooklyn, N.Y. 11203, United States
 SOURCE: Fertility and Sterility, (1980) 33/4 (351-359).
 CODEN: FESTAS
 COUNTRY: United States
 DOCUMENT TYPE: Journal
 FILE SEGMENT: 037 Drug Literature Index
 010 Obstetrics and Gynecology
 004 Microbiology
 LANGUAGE: English

AB In several of the reports the highest conception rates were obtained in couples with unexplained infertility treated with appropriate antibiotics following a positive ureaplasma culture. The great variability in the pregnancy results may be due to a multifactorial etiology for couples considered to have 'unexplained infertility'. A prominent role for ureaplasmas in infertility should not be expected, although some of the data are quite suggestive. A number of investigators firmly believe that ureaplasmas are of importance in infertility, whereas others do not share this view. Genital ureaplasmas have been demonstrated in a large proportion of fertile couples and therefore it has been proposed that only specific strains of urea-plasmas might be causing infertility, perhaps by secretion of specific substances such as **neuraminidase**, ammonia,

or other 'toxic factors' that may **inhibit** conception and/or disturb development of the embryo with the risk of subsequent abortion. Some ureaplasmas in the female genital tract may also adversely affect the function of the tubal epithelium with destruction of the cilia. Serotyping of ureaplasmas might give additional information, but it has also been suggested that the **infection** is only superficial and a systemic antibody response might not be indicative of a current **infection**. For years it has been thought that **mycoplasmas** and ureaplasmas were species-specific and therefore Koch's postulates about an infecting organism could not be carried out. However, it has been recently demonstrated that the chimpanzee can be infected with human ureaplasmas. It is also possible that other, less expensive, subhuman primates can be similarly inoculated. The development of a suitable animal model could provide a valuable new approach for the study of ureaplasmas in human infertility.

L12 ANSWER 4 OF 8 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation. on STN
 ACCESSION NUMBER: 1977:228730 BIOSIS
 DOCUMENT NUMBER: PREV197764051094; BA64:51094
 TITLE: DIFFERENCES IN THE ATTACHMENT OF **MYCOPLASMA**
 -PNEUMONIAE CELLS AND MEMBRANES TO TRACHEAL EPITHELIUM.
 AUTHOR(S): GABRIDGE M G; BARDEN-STAHLE Y D; POLISKY R B; ENGELHARDT J A
 SOURCE: Infection and Immunity, (1977) Vol. 16, No. 3, pp. 766-772.
 CODEN: INFIBR. ISSN: 0019-9567.
 DOCUMENT TYPE: Article
 FILE SEGMENT: BA
 LANGUAGE: Unavailable

AB Hamster trachea organ cultures were exposed to isolated membranes of *M. pneumoniae*, PI 1428. Attachment, monitored by the uptake of tritiated membranes, was relatively insensitive to **neuraminidase** pretreatment, unlike the attachment of viable cells. Membrane attachment was optimal when explants were incubated with 50-100 µg of membrane protein/ml in minimal essential medium broth while gently being rotated (1 rpm) in a roller apparatus for 90-120 min at 37° C. Saturation of the receptor sites with viable cells failed to **inhibit** subsequent membrane attachment. Induction of squamous metaplasia by extended cultivation of tracheal explants in a vitamin A-free medium reduced the content of ciliated cells without significantly affecting total cell viability, but did not alter the attachment of *M. pneumoniae* membranes. Collectively, the data indicate that the mechanism of attachment of *M. pneumoniae* membranes to respiratory epithelium is distinct from the receptor site-mediated attachment of *M. pneumoniae* cells.

L12 ANSWER 5 OF 8 BIOTECHDS COPYRIGHT 2005 THE THOMSON CORP. on STN
 ACCESSION NUMBER: 2004-00309 BIOTECHDS
 TITLE: Use of an agent that **prevents or inhibits Mycoplasma infection**, for manufacturing a medicament for treating or preventing a disorder associated with increased cell proliferation, e.g. atherosclerotic vascular **disease** or malignancy;
 recombinant *Trypanosoma cruzi* protein application in **infection**, tumor and vascular **disease** therapy
 AUTHOR: HIGUCHI M D L; SCHENKMAN S
 PATENT ASSIGNEE: HIGUCHI M D L; SCHENKMAN S
 PATENT INFO: US 2003124109 3 Jul 2003
 APPLICATION INFO: US 2002-86913 1 Mar 2002
 PRIORITY INFO: BR 2001-2648 3 Jul 2001; BR 2000-2989 3 Jul 2000
 DOCUMENT TYPE: Patent
 LANGUAGE: English
 OTHER SOURCE: WPI: 2003-810968 [76]
 AB DERWENT ABSTRACT:
 NOVELTY - Use of an agent that **prevents or inhibits**

Mycoplasma infection for manufacturing a medicament for treating a disorder associated with increased cell proliferation.

DETAILED DESCRIPTION - An INDEPENDENT CLAIM is included for a composition for treating or preventing **Mycoplasma infection** in a subject suffering from a disorder associated with increased cell proliferation or a co-infection with **mycoplasma** and a second microbe, comprising an agent that **prevents** or **inhibits** sialic acid-mediated attachment of **mycoplasma** to cells of the subject.

BIOTECHNOLOGY - Preferred Composition: The agent is an antibiotic or an enzyme having an activity consisting of **neuraminidase** and/or **trans-sialidase** activity. The enzyme is derived from a *Trypanosoma cruzi* microorganism, where the enzyme is a native or a recombinant enzyme. The enzyme has a fully defined sequence of 669 amino acids given in the specification. A vector containing the DNA insert having a fully defined sequence of 2010 bp given in the specification produces the enzyme.

ACTIVITY - Antibacterial; Antiarteriosclerotic; Cytostatic; Anti-HIV. A 64-year-old female patient with a palpable abdominal mass and a tumoral mass in the rectum was administered 50 ml of native **trans-sialidase** (TSN) intraperitoneally on alternate days for a period of 14 days. On day 23, with **mycoplasmas** confirmed in the bone marrow, erythromycin (500 mg/day) was given for a further 20 days. Clinical improvement and normalization of blood leukocytes was seen after 2 days. Considering the important clinical improvement and reduction in abdominal mass, a second session of TSN was administered. The patient demonstrated improvement in general clinical status. Tomography detected a reduction in tumoral mass. Results showed that **trans-sialidase** is effective as a drug in the treatment of neoplasia, removing **mycoplasmas** from the neoplastic cells leading to their apoptosis.

MECHANISM OF ACTION - **Neuraminidase**; **Trans-sialidase**.

USE - The composition or the agent that **prevents** or **inhibits mycoplasma infection** is useful for manufacturing a medicament for treating or preventing a disorder associated with increased cell proliferation, e.g. atherosclerotic vascular **disease** or malignant **disease**, or a **disease** associated with co-infection with **mycoplasma** and a second microbe such as human immunodeficiency virus or a *Chlamydia* microbe (all claimed).

ADMINISTRATION - The amount of the enzyme administered is about 106-1013 units per day. Administration may be intravenous, intraperitoneal, intrathecal, oral, by inhalation, subcutaneous, or intramuscular. (32 pages)

L12 ANSWER 6 OF 8 BIOTECHDS COPYRIGHT 2005 THE THOMSON CORP. on STN

ACCESSION NUMBER: 2003-27952 BIOTECHDS

TITLE: Composition useful for treating **mycoplasma infection** comprises an agent that **prevents** proliferation of **mycoplasma** or associated microbes; native or recombinant enzyme treatment for **disease** therapy

AUTHOR: HIGUCHI M D L

PATENT ASSIGNEE: HIGUCHI M D L

PATENT INFO: WO 2003082324 9 Oct 2003

APPLICATION INFO: WO 2003-BR49 28 Mar 2003

PRIORITY INFO: BR 2002-1010 28 Mar 2002; BR 2002-1010 28 Mar 2002

DOCUMENT TYPE: Patent

LANGUAGE: English

OTHER SOURCE: WPI: 2003-803968 [75]

AB DERWENT ABSTRACT:

NOVELTY - A composition comprises an agent (A) that **prevents** or **inhibits** the proliferation of at least one of **Mycoplasma** or microbes associated with **Mycoplasma**, is new.

DETAILED DESCRIPTION - An INDEPENDENT CLAIM is included for the use of an agent (A) for the manufacture of a medicament for treating a disorder defined by increased microbes proliferation associated with inflammation, fibrosis, calcification, ossification, cellular disarray and/or fragmentation of the extra-cellular matrix of the adjacent tissue.

ACTIVITY - Antimicrobial; Antibacterial; Antiinflammatory; Nephrotropic; Hepatotropic; Endocrine-Gen.; Cytostatic; Osteopathic; Antiarthritic; Antirheumatic; Gastrointestinal-Gen.; Cerebroprotective; Neuroprotective; Antiallergic; Vasotropic; Antiulcer; Respiratory-Gen.; Antiasthmatic; Virucide; Anti-HIV; Dermatological.

MECHANISM OF ACTION - **Mycoplasma** proliferation inhibitor; **Mycoplasma**-associated microbes proliferation inhibitor; Host cell proliferation inhibitor; Microbial proliferation inhibitor. Two rats presenting skin ulcer and tail injury due to the co-infection of **Lycoplasma** and **Spirochetes** were treated. One received 0.5 ml/animal TSN (complete active native trans-**sialidase** of *Trypanosoma cruzi*), every day for 10 days, and the other received TSC (active trans-**sialidase** substance catalytic portion, produced by a recombinant bacteria containing the *Plasmodium* (pTSIII), ATCC with PTA - 3483) for 8 days. The mice were killed respectively with 14 and 10 days. The skin ulcers already showed initial healing after 4 days of treatment, with complete healing in 14 days, with the formation of a new coat. There was a stop in the loss of the tail and the histological exam demonstrated regression of the lesion and severe decrease of all infectious agents.

USE - For treating or preventing **Mycoplasma** infection including disorders defined by co-infection and fusion of **Mycoplasma** and/or at least a second microbe to a host cell or a cell fragment, causing inflammation and at least one of the tissue alterations due to fibrosis, calcification, ossification, cellular disarray or fragmentation of the extra-cellular matrix of the subjacent tissue (e.g. aortic valve stenosis with calcification, idiopathic glomerulopathy, glomerulopathy with inflammation, Lyme's disease, co-infection with chlamydia, spirochete and/or archaea); and for the manufacture of a medicament for treating a disorder defined by increased microbes proliferation (e.g. calcification of the cardiac valves, glomerulonephritis, fibrosing chronic hepatopathy, baldness, and malignant neoplasia) (claimed). Also useful for the treatment of skin ulcer, osteoarthritis, inflammatory bowel disease, chronic cerebral sclerosis disease, lymphocytic chronic arteritis, non-purulent inflammatory osteoarthritis, multiple sclerosis, lymphocytic inflammatory vascular disease, optionally granulomatous and with non-stabilized etiology (e.g. Takayasu's disease, giant cell arteritis, Wegener's granulomatosis, thromboangiitis obliterans), rheumatoid arthritis, ulcerative colitis, Whipple's disease, gastritis, inflammatory diseases of the respiratory tract of not well established etiology (e.g. adult respiratory distress syndrome, Goodpasture's syndrome, asthma, chronic fibrosing hepatopathy, emphysema; and for the treatment or prevention of disorders associated with **mycoplasma** infection, co-infection and/or fusion of **mycoplasma** with other microbes (e.g. virus such as human immunodeficiency virus, hepatitis virus, cytomegalovirus, human papillomavirus, Epstein-Barr virus; or bacteria).

ADMINISTRATION - The trans-**sialidase** enzyme is administered in a dosage of (4 mg/day) in a period of at least 2, or a culture of *Trypanosoma cruzi* with a mean trans-**sialidase** activity of 140 U/day is administered every other day for one week (1 - 8 weeks). The administration is intravenous, intraperitoneal, intrathecal, oral, by inhalation, subcutaneous or intramuscular.

ADVANTAGE - The composition inhibits or prevents the adhesion and/or infection of **Mycoplasma** and the microorganisms associated with them by at least 10%. The antibiotic protein such as **neuraminidase** enzyme or the trans-**sialidase** enzyme of *Trypanosoma cruzi* removes the sialic acid

residues and **inhibits** or **prevents** the attachment of **Mycoplasma** to host cells.

EXAMPLE - No relevant example given. (24 pages)

L12 ANSWER 7 OF 8 BIOTECHDS COPYRIGHT 2005 THE THOMSON CORP. on STN

ACCESSION NUMBER: 2002-08674 BIOTECHDS

TITLE: Composition useful for treatment of **mycoplasma infection** and **diseases** associated with cell proliferation e.g. malignancy or with co-**infection** with another microbe, comprises agent inhibiting sialic acid-mediated attachment of **mycoplasma**; native or recombinant enzyme treatment and vector-mediated gene transfer and expression in host cell for **disease** therapy or prevention

AUTHOR: HIGUCHI M D L; SCHENKMAN S

PATENT ASSIGNEE: HIGUCHI M D L; SCHENKMAN S

PATENT INFO: WO 2002002050 10 Jan 2002

APPLICATION INFO: WO 2000-BR83 3 Jul 2000

PRIORITY INFO: BR 2000-2989 3 Jul 2000

DOCUMENT TYPE: Patent

LANGUAGE: English

OTHER SOURCE: WPI: 2002-154675 [20]

AB DERWENT ABSTRACT:

NOVELTY - A composition useful for treating or preventing **mycoplasma infection** in a subject suffering from a disorder characterized by increased cell proliferation or by co-**infection** with a second microbe comprising an agent that **prevents** or **inhibits** sialic acid-mediated attachment of **mycoplasma** to the subject's cells, is new.

DETAILED DESCRIPTION - An INDEPENDENT CLAIM is also included for use of an agent preventing/inhibiting **mycoplasma infection** in medicaments to treat disorders characterized by increase cell proliferation.

BIOTECHNOLOGY - Preferred Composition: The agent is preferably an enzyme (native or recombinant) with **neuraminidase** and/or trans-**sialidase** activity, especially derived from *Trypanosoma cruzi*. It preferably has fully defined sequence (I) of 669 amino acids as given in the specification. The medicament preferably includes a vector comprising DNA insert of a fully defined sequence (II) of 2010 base pairs as given in the specification, producing the preferred enzyme having sequence (I) as above.

ACTIVITY - Antiatherosclerotic; antibacterial; antiviral; anti-HIV; cytostatic; vasotropic. A laboratory rat population was determined to be infected with both **Mycoplasma pulmonis** and *Chlamydia pneumoniae* using standard immunohistological techniques and histological examination of various organs. Nine adult rats (approximately 285 g; seven males, two females) presenting conjunctivitis, slow movements and, in one rat severe otitis and another severe weight loss, were allocated to groups A or B. A (Control group) comprised three animals, two of which were killed without injecting any substance and one receiving an inactivated form of 'TSC' (recombinantly produced catalytic portion of *Trypanosoma cruzi* trans-**sialidase**) for 5 consecutive days. B comprised five animals receiving 'TNS' (complete, native *Trypanosoma cruzi* trans-**sialidase**) (0.5 ml/animal, every 2 days) and sacrificed after 7, 9, and 12 days, and one rat receiving active TSC (140 microgram/day, five consecutive days) and killed after 7 days. Group B rats showed a clear improvement in symptoms, becoming more agile, requiring more ether to anesthetize them and becoming more difficult to restrain. The animal with otitis showed less equilibrium loss and the animal with severe weight loss gained weight. Since the lung was the most frequently injured organ, pulmonary alterations were examined using electron microscopy, confocal laser microscopy and immunohistochemistry. Histological sections showed that treated animals presented resolving pneumonitis after 7 d. After 9-12 days *M. pulmonis* were almost absent from alveoli and mean C.

pneumoniae positive cell numbers in alveoli had decreased, compatible with regression of C. pneumoniae **infection**. Results are given in the specification.

MECHANISM OF ACTION - **Inhibits** sialic acid mediated attachment of **mycoplasma** to cells.

USE - The compositions are useful to treat **diseases** associated with undesirable cell proliferation, such as atherosclerotic vascular **disease** and malignancy (both claimed), by reducing or preventing **mycoplasma infection**. They also useful to treat **diseases** associated with **infection** with other infectious organisms co-occurring with **mycoplasma** (and typically increasing the virulence of both pathogens), especially human immunodeficiency virus or chlamydia species. They can be used to treat such **diseases** in humans or other animals, and can be administered in conjunction with conventional agents e.g. anti-platelet or chemotherapeutic agents. (63 pages)

L12 ANSWER 8 OF 8 HCAPLUS COPYRIGHT 2005 ACS on STN

ACCESSION NUMBER: 2003:261602 HCAPLUS

DOCUMENT NUMBER: 138:265609

TITLE: Use of **neuraminidase** inhibitors to **prevent** flu-associated bacterial **infections**

INVENTOR(S): McCullers, Jonathan A.

PATENT ASSIGNEE(S): St. Jude Children's Research Hospital, Inc., USA

SOURCE: PCT Int. Appl., 40 pp.

CODEN: PIXXD2

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2003026567	A2	20030403	WO 2002-US29417	20020917
WO 2003026567	A3	20040826		
W:	AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW			
RW:	GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG			

US 2004248825 A1 20041209 US 2004-809127 20040325

PRIORITY APPLN. INFO.: US 2001-325615P P 20010927

WO 2002-US29417 A1 20020917

AB The invention provides a novel use for **neuraminidase** inhibitors in chemoprophylactic and treatment methods for the prevention, attenuation, and treatment of bacterial **infections** that may occur in association with, or as a sequelae of, viral influenza. The prophylactic methods of the invention are particularly suitable for the prevention of secondary bacterial **infections**, such as, but not limited to, **infections** of the lower respiratory tract (e.g., pneumonia), middle ear **infections** (e.g., otitis media), and bacterial sinusitis. The treatment methods are suitable for use in protocols designed to attenuate or treat bacterial **infections** that occur concurrent with, or as a sequelae of, the flu.

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(FILE 'HOME' ENTERED AT 14:05:18 ON 10 JAN 2005)

FILE 'MEDLINE, EMBASE, BIOSIS, BIOTECHDS, SCISEARCH, HCAPLUS, NTIS, LIFESCI' ENTERED AT 14:05:42 ON 10 JAN 2005

L1 73150 S MYCOPLASMA
L2 63554 S NEURAMINIDASE? OR SIALIDASE?
L3 224 S L1 AND L2
L4 139 S (INFECTION? OR DISEASE?) AND L3
L5 2620647 S CANCER OR "HODGKIN'S" AND L4
L6 87 DUP REM L4 (52 DUPLICATES REMOVED)
L7 652 S (CANCER OR "HODGKIN'S") AND L6
E HIGUCHI M/AU
L8 3027 S E3
E SCHENKMAN S/AU
L9 344 S E3
L10 3371 S L8 OR L9
L11 2 S L6 AND L10
L12 8 S L6 AND (PREVENT OR INHIBIT)